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THE PHILOSOPHY OF MENINGITIS.*

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The "why" of meningitis: why one patient gets it and another doesn't; why it kills. An understanding of these "whys" contains the key to *how* to guide the patient to recovery.

Classification of Types of Meningitis: There is a prevailing lack of understanding of meningitis in general, chiefly because of improper nomenclature. Our classification is based on bacteriology and termination. Thus, we say pneumococcus meningitis,—all cases of pneumococcus meningitis die; or we say meningicoccus meningitis,—meningicoccus meningitis cases recover.

The classification is also based on the *location of the primary focus;* thus we speak of meningitis of aural or nasal origin, and believe that all meningitis cases resulting from ear or nose disease, die. When now and then one does recover, we complacently say it was not a true meningitis, but a case of meningismus. This large group of meningitis cases which are preceded by or associated with both ear and nasal mucous membrane suppuration (and this evidently is a manifestation of a general systemic state), we dismiss as of "bloodstream origin", or more vaguely still, satisfy our ignorance by applying to such cases the term "concomitant meningitis". Concomitant is a good example of the tendency of the human mind to dodge the issue by the use of a name.

Language is the means whereby the individual expresses himself to others. Language should stimulate thought, and when properly

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used, it does so. A famous English statesman, on being asked when he had time to think, replied: "Think, think? I never think unless I have to put it on paper." Language can also be used to hinder thought, and too often we find an excuse for the continuation of ignorance by giving a name to something which we do not understand, the very naming of which hinders investigation, because our curiosity is satisfied by the name. "Concomitant" is one of these terms. Concomitant meningitis means a meningitis which is concomitant with something else, and as we know nothing much about that something else, we are apt to infer in a vague sort of way that because of the diversity of the lesions, a bloodstream infection must exist and is the origin of the meningitis. All this confusion and lack of understanding is due to a failure to grasp a few simple principles of general pathology and anatomy.

In my office I have three dictaphones. The dictaphone's chief value to me is not in the rapid recording of my observations at the time of the examination of the patient, although this is important; its great value is that it makes me commit myself to a definite opinion; it does not allow me to dodge the question, "Do I know what is the matter with the patient? Do I not know? If I do not know, what can it be? *Why* is it probably this or that?" It makes me face the issue.

What is meningitis? Why does it occur? Why is it so generally fatal? How can it be cured?

I am going to try to answer some of these questions in the light of present knowledge and of my own experience, for I have been studying its treatment and trying to understand meningitis for several years. In every case that I have treated I have tried to find out why the patient died, or tried to explain why he recovered. In doing this I have made a sincere effort to obtain a postmortem in all cases that died, and I have succeeded in a very large number, so that my views are largely based on clinical notes and postmortem observations.

Present Knowledge Concerning Meningitis: First, in studying the literature I have been surprised to find that among the thousands of cases of postmortems, the number which has been subjected to *microscopical examinations* to trace the infection into the meninges is very few indeed. In fact, the total number can almost be counted on the fingers of your two hands; it does not exceed 12.

Second, that nearly all our knowledge about the real *causes* of meningitis has been obtained from animal experimentation.

Third, that much more is really known about the group of diseases usually described under the heading of meningitis than is generally conceded; but these facts are isolated and disconnected. The purpose of this address is to correlate these known facts with my individual experience.

Regional Approach to Study of Meningitis: In the first place, I am persuaded that meningitis, to be properly understood and surgically treated, must be considered in much the same way as we now approach cancer. We know that an epithelioma of the skin of the face is an entirely different thing from an epithelioma of the mucous membrane of the mouth. In this same way we should not say "meningitis of nasal or aural origin", for we know clinically that a suppurative meningitis which goes in through the cribriform plate of the nose has an entirely different course than one which enters from the frontal sinus or the ethmoids, although these structures are contiguous with each other. A case of traumatic fracture which goes across the cribriform plate frequently recovers without meningitis,—in fact, few die, but those operated on the nose with a fracture of this plate rarely recover; practically all such cases die of meningitis. Again, a meningitis that goes in by way of the labyrinth has an entirely different clinical course from a meningitis which originates from caries of the cells of the mastoid.

Clinical Course of Meningitis: This difference in the clinical course between the meningitis from cribriform plate fracture and meningitis from frontal sinus infection—between that from labyrinth suppuration and mastoid cell disease—is *regulated by the differences in the structure of the arachnoid tissue* which are first encountered as the infection advances from without through the perineural spaces of the cribriform plate, or by the veins of the frontal sinus. There is a different clinical course, depending upon whether the aural suppuration goes by way of the bone of the mastoid cells or through the endolymph of the labyrinth. Consequently to understand these clinical differences we must not only have an understanding of the exact physiological and anatomical route of invasion into the subarachnoid, but also of its course in the arachnoid. And if we do understand the minute anatomy and the physiology of the overlying structure; be it bone, mucous membrane or dura—and the exact anatomy and physiology of the subarachnoid spaces and cerebral tissue, then we will be in a better position to diagnose suppurative meningitis at an early date before it becomes a general leptomeningitis, and also to combat surgically the localized infection.

Protective Mechanism of the Meninges: Meningitis is a disease engrafted onto the specific connective tissue coverings of the brain.

Life, in its evolution, has covered the central nervous system *1. with bone—the skull* and *2. with a firm envelope—the dura.* The *bone* is adapted to protect the delicate nervous tissue from gross external violence, blows, wounds, et cetera; the *dura* to protect it against microscopical disturbances,—infection, variations in temperatures, atmospheric pressures and what not.

The surgeon can mutilate, hammer and tear, freeze or infect the *tissues of the skin and the skull* to his heart's content, all under the legalized term of operation, and the sheltering hood of misdirected medical ethics, *provided* that all this trauma and abuse are confined to the tissues external to the inner surface of the dura.

Life has gone still further, for *inside the dura* it has placed a framework analagous to, but different from, the lymphatic system; a framework which is necessary for the proper functioning of the cerebral tissue which it envelops and permeates. This supporting framework — the pia-arachnoid — also constitutes a barrier which stands between the cerebral tissue and all extrinsic influences,—for although of microscopical consistency, it says to every corpuscle, every drop of blood serum, every bacterium, and even most toxemias, "Thou shalt not pass."

But life has gone further than this, for this framework is not only a barrier, but also contains a *protective mechanism,* so that any infection which may pass the barrier of the dural covering, or which may be brought to the brain by the circulatory blood, will be instantly counteracted, digested and removed.

If we are to understand meningitis we must understand this framework, this barrier, this protective mechanism, this phagocytic membrane, not only in its gross anatomy, but also in its microscopical physiology and especially its reactions in the presence of an irritant, —be it trauma, cold or micro-organisms.

This framework, this *nutritional system,* this barrier, this *protective mechanism,*—the cerebrospinal fluid system,—varies greatly in its anatomical peculiarities, depending *1.* upon the part of the brain it covers, *2.* the physiology of the overlying anatomical tissues, and *3.* the intimacy of its development with the overlying structures.

There is a common source of development of the bone, the dura and the arachnoid cells; they are all specializations of connective tissue. Life has evolved them for one purpose,—to protect the ganglionic tissue of the central nerve tissues.

Wherever we find a center of ossification in the skull we find an adherent dura; wherever we find an opening in the dura for the

exit of nerves or veins, or the entrance of arteries, we find an arachnoid prolongation. We can go inward, *into the brain itself*, and still find this intimate developmental continuity, for if we dissect the injected arteries of the brain we will find that the arachnoid prolongations follow them in all their ramifications, the arachnoid meshes apparently springing from the arteries—which in fact they do; for the arteries, veins and arachnoid of the brain are *all* specialized connective tissues, and were evolved simultaneously.

Constructing Model of the Arachnoid Meshes: Consequently if we know the course of the blood vessels entering the brain we can construct a working model of the arachnoid meshes. The larger the artery, the thicker the mesh of arachnoid; the smaller the artery, the more delicate the perivascular space. We can go still further in the construction of our model of the arachnoid, for if we follow each nerve and vein, be it present or only an embryological remains of one, as it leaves the skull we find it accompanied by an arachnoid prolongation. In meningitis it is *these extensions* of the pia-arachnoid externally that interest us—the arachnoid prolongations which surround the nerves and veins as they leave the brain, and pass out into the overlying structures, because as they go out into the bone they are apt to become the avenues by which infection of the surrounding bone or of the accompanying veins or nerves may get into the subarachnoid tissues. These arachnoid prolongations therefore become points of *especial vulnerability* to infection, depending upon the liability of the overlying structures to infection.

Points of Vulnerability: Anatomically, then, as well as clinically, the pia-arachnoid presents three main points of vulnerability to infection—the *nose*, the *ear* and the *bloodstream*—because these are points at which the arachnoid spaces are closest to the tissue that is frequently infected.

Of course any accident which opens the subarachnoid spaces makes a meningitis possible. Thus, a compound depressed fracture of the vault of the skull, if followed by a high temperature, stiff neck, headache and swelling over the parts,—and especially if accompanied by a cloudy cerebrospinal fluid from the lumbar region,—would cause any general surgeon to follow the path of infection by removing the depressed fracture, and if under the area of infected bony depression, there is found a large amount of cerebrospinal fluid, its evacuation is often followed by the recovery of the patient.

Why not try this principle in meningitis from the ear or nose, instead of saying pneumococcus or streptococcus meningitis? Why not try to diagnose how the infection reached the arachnoid, as is

shown in the case of the depressed fracture? To do this we must understand not only the differences between the various overlying structures, but also the mechanism which life has constructed in different locations for its own protection, some being adapted to combat infection, others being adapted to resist trauma (G. Elliot Smith: The Evolution of Man. Oxford Univ. Press, 1927).

Difference Between the Nose and the Ear: The nervous mechanism of the nose has been evolved for the continuance, propagation and defense of the primitive animal. (The lower down in the scale of life we go, the more its olfactory mechanism dominates its existence.) This smell sense, which in the higher animals is of but secondary importance as far as existence is concerned, but which is now devoted largely to higher functions, occupies an exposed place in the anterior part of the head. It is exposed to constant infection from the air, and is surrounded by a specific mechanism for the control of infection—the mucous membrane of the nose. Because of this the olfactory portion of the brain is fully protected from infection, although the bone covering it is *a.* thin, *b.* perforated, and *c,* the arachnoid prolongations surrounding it actually leave the skull for a considerable distance. Thus, infection of the nasal mucous membrane, which is adapted to control by its protective mechanism, rarely causes meningitis. However, the cribriform plate does not tolerate trauma. If the prolongations are entered, a rhinorrhea persists until the death of the patient from meningitis.

On the other hand, the middle ear and mastoid cells, which have no such protective mechanism—and thus are especially liable to infection—if fractured, rarely cause suppurative meningitis; the cerebrospinal fluid leaks for a few hours or days and then stops.

Meningitis occurs in only *about 8 per cent of the untreated* cases of fracture of the temporal bone through the dura, but in nearly 25 per cent of the untreated cases of fracture through accessory nasal sinus in which the dura is perforated. This is because of the difference in the physiological structure of the two overlying regions, one being constructed to resist infection, the other, trauma.

Infection in General: Let us look at infection in general, and especially at the clinical differences of its course in relation to the different tissues of the body. A surgeon incises a boil on the neck; nothing comes out. He squeezes it, and beyond making the patient more uncomfortable than he was before, nothing happens. The surgeon, being a scientific animal and, being scientific, requiring a written report from another animal who spends his life in a distant building looking through a microscope and never at a patient,—as if life were a disjointed affair—is solemnly informed that "the cul-

ture from the blood received at the laboratory shows staphylococcus". The surgeon says, "Oh, staphylococcus never kills". But the swelling on the neck having by this time become much more extensive, the surgeon (if the patient will allow him—which he usually will not) proceeds to cut him again, and now the surgeon says the boil is a carbuncle which needs many cuttings.

But the patient has a boil on the lip or inside the nose. It is red, brawny, has a bluish hue and is painful. The surgeon cuts it deeply. It cuts like liver, but as nothing comes out, the surgeon, being sure that there is infection, squeezes the swelling; nothing but blood escapes, and the laboratory animal again reports staphylococcus. The next day the patient has a chill; then one eye protrudes and the patient is delirious and soon dies of cavernous sinus thrombosis. Then the surgeon learns that staphylococcus on the back of the neck and staphylococcus on the lip apparently act differently. After killing a few patients, that surgeon even learns not to squeeze boils on the lip, but his faith in the necessity for steel in infection never weakens. Is it not a dictum that infection requires drainage? The knife must follow infection as civilization follows the flag, although no knife ever cured a case of erysipelas, and the backward races are soon extinguished by syphilis, drunkenness and in poverty.

A surgeon excises the skin and bone of the face or mastoid. If the knife is infected, he has a suppurating wound. If he cuts the mucous membrane of the nose with the same dirty knife, nothing untoward happens. But now and then when there are pneumococci in the tissue of the nose or on the forceps, a fulminating meningitis kills the patient in 48 hours.

If during a frontal sinus operation the dura is accidently cut and cerebrospinal fluid escapes, meningitis rarely occurs, for the protective mechanism controls it; but if in a fracture of the frontal sinus a piece of bone is driven into the subdural space, the first cold in his head that the patient contracts is followed by a meningitis, although he may not have any symptoms for a long time.

During a mastoid operation if the surgeon perforates the sinus with his knife, nothing happens; but if he traumatizes the inner sinus wall, he is apt to have a meningitis.

Experience teaches that in performing a radical operation for chronic suppuration, if the surgeon injures the outer arm of the horizontal semicircular canal cerebrospinal fluid leaks for a few days or weeks, then stops, and except for the symptoms caused by the sudden removal of labyrinth stimuli, nothing happens. However, if he dislocates the stapes into the vestibule (unless the suppuration is prevented by a prompt surgical destruction of the labyrinth) the

patient dies of meningitis, although the arachnoid spaces behind the petrous pyramid can be opened with little danger of infection. In the case of the dislocated stapes, he has opened a cisterna at a place where it cannot be obliterated, and has left an infected foreign body in it to *continue the infection,* but in incision of the dura the pia-arachnoid soon obliterates the infected area by adhesions.

Consequently whether infection occurs in the meninges themselves it is a question of *1.* the kind of tissue through which the infection enters, that is, the route of the infection; *2.* if a protective mechanism is ready to control it at the particular place, that is, the reaction of the tissues to the irritant; as well as *3.* the virulence of the infection; and *4.* the *degree of the trauma* inflicted by the infection or the operation.

Reaction of Meninges to Irritants: Adami said that all inflammation is a reaction to an irritant. Let us look at the reaction of the meninges to different irritants.

1. Nonbacterial Irritants: a. Cold. If a stream of water is passed through the subarachnoid spaces at a temperature of but little below 100° F., respiration ceases. *b. Chemicals.* If sterile water without a small calcium content is passed through the arachnoid meshes, there is apt to be a rise of temperature to 105° F. or over; if we run a drug, such as mercurochrome, even in a small amount, *through* the spaces, respiration stops, and unless artificial respiration is continued for some time, the animal dies.

The laboratory terms this drug death, and the laboratory has learned to keep up artificial respiration as long as the animal's heart beats, which may be for some time. All surgeons using mercurochrome in the spine or brain should remember to remain with the patient for some time, and if the breathing stops, to keep up artificial respiration.

Case 1: In a case of general suppurative meningitis of aural origin, a subarachnoid irrigation with properly constituted watery fluid from the frontal region into the occipitoatloid and lumbar regions had no appreciable deleterious effect on the pulse and respiration. But while the needle was still in place, injection by gravity into the cisterna magna of a serum at the temperature of the room (about 72° F.) was immediately followed by respiratory failure and cardiac embarrassment,—shallow and infrequent respiration, rapid, thready pulse, cold, clammy sweat,—which continued for at least 15 minutes.

Dryness. If the arachnoid cells are exposed to the air for but a few minutes, they die. From this we learn that in any inflammation of the arachnoid, our therapeutic agent must be warm, must be properly constituted, and must prevent dryness.

1. Autogenous Irritants. We have an irritant which, *while always present* near the subarachnoid spaces normally, *is never in them*—that is, *blood*—free blood from the patient himself. If we heed the differences in the clinical manifestations which follow blood effused into the subarachnoid spaces of the frontal region and over the brain stem, we will get an idea of the tremendous importance of different locations when irritated by the same irritant.

Blood when transfused into the frontal region does not appear to cause any marked symptoms of cortical irritation. Fractures of the anterior fossa rarely give signs of irritation. Frontal lobe abscesses seldom give outstanding symptoms until they are so large as to cause general compression; but *free blood over the bulb* causes stiff neck, high temperature and symptoms of cerebral intoxication.

Thus we have a marked manifestation in subarachnoid hemorrhages in the region of the brain stem, but it is entirely absent when the blood is effused into the frontal region.

2. Chronic Infections. Syphilis. The organism of syphilis apparently has a marked predilection for attacking the frontal region of the pia-arachnoid, as is evidenced by the nocturnal frontal headache, the blindness from optic atrophy of tabes, and the psychic symptoms of general paresis.

From all this we may learn that vegetable and animal irritants, like blood, all act differently in different parts of the arachnoid tissue, and have certain predilections for certain areas.

When we come to the bacterial irritants we find that the piarachnoid may be infected by all kinds of micro-organisms, from the self-limited organism—meningococcus—to the pneumococcus, which, although it is self-limited in the lung—for the *pulmonary tissue eliminates the micro-organism by resolution,* but produces no such limitation in the arachnoid.

The arachnoid exhibits all degrees of infection, from the chronic infection of tubercle bacilli localizing in tuberculosis (from tuberculous meningitis and death after weeks or months), to the highly virulent encapsulated pneumococcus, with death within a few hours; but each type and kind of micro-organism shows *certain peculiarities* and *characteristics,* depending upon how it *enters and extends* into the arachnoid. Some of these differences we know, and of others we have little idea.

Thus the meningococcus apparently has a predilection for the frontal region, and there is some evidence that the arachnoid prolongation that perforates the cribriform plate is the route by which this micro-organism reaches the subarachnoid spaces. On the other

hand, whether this specific self-limited micro-organism spreads from here into the bloodstream or *vice versa* is as yet undetermined.

We can definitely say of the meningococcus *1.* that it is *self-limited,* as shown by the rapid disappearance of the micro-organism from the fluid.

2. It is *in* the bloodstream, as is shown by the purulent chorioiditis that so frequently occurs.

3. It causes *exudate,* as is shown by the total deafness.

4. It is amenable to a specific serum, which should be given intracisternally and intravenously.

But there are other things about meningococcus, in addition to its being in the bloodstream, that are not as yet well understood—such as its relation to nasal sinus disease. It has been demonstrated that infection of the accessory sinuses of the nose (sphenoids and posterior ethmoids), by pathogenic micro-organisms (streptococci) is a frequent accompaniment of meningococcus disease of the pia-arachnoid, and Edleson has demonstrated that the surgical opening and drainage of a sphenoidal empyema facilitates the cure of meningococcus meningitis in those cases which persist in spite of serum treatment.

So here we have a bloodstream infection with a specific type of meningitis which is *self-limited in the meninges,* aggravated or possibly kept going when another pathogenic organism's presence is confined in an accessory nasal sinus.

Control of Infection by the Arachnoid: But before the death of the patient, while the meningococcus is still present — while the depressed piece of bone of the frontal fracture still projects into the dural tear—what effort is the arachnoid making to control the infection already within it? Any postmortem examination of a case that died of meningitis will show that when any micro-organism, no matter how virulent, is engrafted on the arachnoid, the arachnoid tries to limit the area infected *1.* by the formation of adhesions of one mesh with another. Thus we have *lakes of infected cerebrospinal fluid* in juxtaposition to the infected area in the ear or nose, and in addition *2.* the arachnoid cells actually *digest* the invading micro-organisms. For these arachnoid cells, these wandering macrophages, these mast-cells, as they are called, are a part of the great protective reticuloendothelial systems given the body for its protection against foreign irritants. So if we examine any case of infection, whether it be from the nose or the ear, we will find first an *effort at localization;* second, *of control.*

If we can but diagnose the exact site at which the infection enters the system,—if we but study the course that it normally takes in that system,—for meningeal infection *follows pathways* exactly as the infection from the finger goes up through the lymphatics to the axilla—if we will but open the localized area of infected fluid early enough; further, if we can but stimulate the cerebrospinal fluid cells to activity by increasing their immunity, and if we will assist the curative efforts by putting the inflamed tissue at rest, then we will get many of these cases well.

Meningitis from the Nose. I have said that the reaction of the piarachnoid barrier, and its efficiency as a protective mechanism, vary greatly, depending upon *1.* its own *neighborhood* formation; *2.* the *overlying tissue and 3.* its arachnoid prolongations, which extend outward into the bone, being vulnerable points of infection. All these points are well shown in cases of meningitis which originate from the nose, for all meningitis originating from the nasal mucous membrane should be divided into two groups, depending upon whether the infection enters the arachnoid along the perineural sheath of the olfactory nerve, through the cribriform plate, or from the bone of the frontal sinus or ethmoids.

Medial and Later Groups of Frontal Lobe Meningitis. A better designation would be *meningitis* from infection of the *1. frontal cisterna,* and *2. localized meningitis of the meshes of the arachnoid under the frontal lobe.* These two groups differ in their course, symptomatology and prognosis because of an anatomical peculiarity, which permits the infection to extend directly into the large cisterna which surrounds the olfactory bulb as it lies over the cribriform plate, or to reach first the piarachnoid on the under surface of the frontal lobe.

The Lateral Group. Infection occurs through the inner wall of the frontal sinus, by erosion of the bone, or through a thrombophlebitis of a small vein, but in every one of these cases the infection is controlled often for a long time by the close mesh which it encounters in the subarachnoid space.

Case 2: A man has a frontal sinus disease. He is operated upon. Some time later he develops headache, rather irregular temperature. A lumbar puncture is performed and there is a high cell count in the cerebrospinal fluid. A large area of bone is now exposed over the frontal region and an orbital prolongation over the frontal sinus containing pus, is discovered. This reaches well out towards the outer portion of the orbit. The bone over the area of pus is removed, underneath which the dura is perforated, and on incising the dura a large quantity of infected cerebrospinal fluid is evacuated.

Case 3: A man has a frontal ethmoidal sinus disease. One day he has intense pain in the head and a stiff neck rapidly develops; he vomits, and a lumbar puncture reveals a high cell count with bacteria in the fluid. The common carotid artery of the same side is ligated, the dura is exposed, a large quantity of cerebrospinal fluid is evacuated, the skin is made to grow in over the opening of the frontal sinus into the dura, and after repeated lumbar punctures, followed by repeated transfusions, the man makes a rapid recovery. Here, then, is a case in which the median line was partially involved.

Case 4:. Medial Group. A surgeon does a submucous resection of the nose; another surgeon opens the sphenoid, and a third surgeon removes the middle turbinate. The following day the patient has a terrific headache, high temperature, stiff neck; and the third day after that, the patient is apt to be dead from general suppurative meningitis.

Why this quick death in one type and the slow course in the other? The infection has gone up the median line through the prolongations of the piarachnoid into the large cerebrospinal fluid lake over the cribriform plate. At the cribriform plate the piarachnoid, dura and bone are, for surgical purposes, all one structure. The piarachnoid cannot wall itself off. Infection of one causes infection of all, and the flow of cerebrospinal fluid is continuous, coming *from each side in the region of the third nerve.* Where the anterior cerebral joins the middle cerebral there is one of the big pathways. Normally this region of the cribriform plate is meant to be bathed in fluid; when infected, the fluid is constantly disseminated over the whole base,—the cisterna, cerebellomedullaris and the sides of the hemispheres by way of the sylvian fissure. The *infection is pumped,* as it were, by the ebb and flow of the current, by the venous aspiration or the arterial impact, whatever may be the force of the cerebrospinal fluid movement. The fluid is pumped into and out of the large lake, and when infected, the infection cannot be limited, but is disseminated.

Rhinorrheal Cases. There is also another group of cases—meningitis of the frontal region; *meningitis which follows a rhinorrhea,* for it has long been known that all people suffering with rhinorrhea ultimately die of meningitis. Why does rhinorrhea occur, and why does meningitis always develop?

Case 5: A man has a fractured skull, the bone over the frontal region being driven into his cerebral tissue. An operation is performed with elevation of the bone, and the man is discharged from the hospital as cured, but with a rhinorrhea. Every now and then

he has a persistent watery discharge from one nostril. Suddenly following a cold in his head he develops a meningitis and dies within 24 hours.

The postmortem examination shows not only *diffuse suppurative meningitis,* but what is apparently a cyst that runs into the cerebral tissue under the lateral ventricle. This cyst communicates with the frontal sinus, which is kept washed out by the discharge of fluid from the contents of the cyst. When the cyst fills with fluid, the fluid discharges from the nose. When it is empty, the discharge stops.

Case 6: I saw another man who had been shot through the frontal sinus and he likewise was discharged as cured from the hospital. Suddenly the same history, a cold in the head, headache, coma, general meningitis and death.

A postmortem shows that he also has what looks like a cyst running from the lateral part of the frontal sinus into the cerebral substance. But macroscopically this cyst is not a cyst. It is an ingrowth of mucous membrane of the frontal sinus through the dural opening into the arachnoid, so that with each cold an infection travels directly through the unclosed opening into the arachnoid tissue, and this postmortem shows the cause of both to be rhinorrhea and death in these cases. The explanation of rhinorrhea, then, is *lack of closure.*

The nasal mucous membrane, being a low-grade tissue, proliferates rapidly. Given an opening from any of the accessory sinuses through the dura, the *mucous membrane proliferates through it.* This opening, being in communication with the general subarachnoid space, a cerebrospinal rhinorrhea results, and infection travels through this path into the subarachnoid spaces, ultimately resulting in the death of the patient.

15 Lombardy Street.

AN UNUSUAL CASE OF LATERAL SINUS THROMBOSIS.*

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First, in presenting this case, I would like to emphasize the unusual onset with practically no symptoms pointing to sinus thrombosis. The ear symptoms were so mild that we had to go into the history very carefully with the parents to develop this fact. Second, the Queckenstedt-Toby test was the outstanding fact in making the diagnosis of lateral sinus thrombosis.

John W., age 11 years, white, home near Philadelphia, admitted to hospital Sept. 5, 1928; died Sept. 27, 1928. Father and mother living and well. Two brothers living and no history of ear conditions in other members of the family.

A previous history shows the child had had scarlet fever, measles, chickenpox and mumps. He was not subject to sore throats. Tonsils and adenoids had not been removed.

The child was perfectly well up until Aug. 1, 1928, when he had an attack of tonsillitis. Was sick around home three or four days, but did not go to bed. Patient gave history of swimming in pool. Seemed to have recovered perfectly and was as active as usual, swimming, etc., when about Aug. 22 had another attack of tonsillitis, with fever and an earache in left ear, lasting for six to eight hours. No swelling back of ears. Was put to bed. Throat very sore and left tonsil red and swollen, but did not seem to bother him very much, other than running a high fever, with daily remissions. One week before admission to the hospital had been running septic temperature, ranging from 99° to 105°. Some swelling noted in neck, but had no trouble swallowing. Bowels opened last two days with enemata. Eating poorly the past four days and did not take fluids very well. Did not vomit until three days before admission. No pain anywhere, no convulsions and no delirium. Had chill Saturday, Sept. 1, 1928, followed by headache. Stools normal throughout—no blood, and since he had been in swimming pool waters, a Widal was taken for typhoid, with negative results.

Physical examination at the time of entrance to the hospital showed the patient a well nourished boy, lying quietly in bed. Did not appear to be seriously ill nor had he any signs of having gone through a

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recent severe illness. No gross abnormalities. Head, negative. Ears, membrana tympani negative, both sides. No tenderness over the mastoid. Eyes, Pupils large, equal, regular and react well to light and accommodation. Mouth, hygiene fair, tongue clean, tonsils large, cryptic and exuding pus. Not acutely inflamed at present. Sinuses, negative to transillumination. Neck, no rigidity, smooth glandular swelling on left side from below the angle of the jaw downwards. Chest, expansion free and equal. No impairment on percussion. Breathing, sounds vesicular. Fremitus, normal. Heart, size normal, rhythm regular. Mitral murmur heard. Abdomen, retracted, no masses, tenderness or rigidity. Peristalsis, normal. Extremities, negative, reflexes normal.

Tentative Diagnosis: Cervical adenitis; septicemia.

Sept. 6, 1928: Patient complained of some pain in neck. Fusiform soft swelling on left side. Slightly tender and temperature up this p. m.

Sept. 8: Dr. Babbit visited the patient and made the following observation. Some fullness and tenderness in cervical area beneath left ear. Tonsil picture could not account for this, but note peculiar septic temperature. His left tympanic membrane is red, with fullness in outer upper quadrant. No tenderness over mastoid antrum. From my standpoint the condition somewhat suggests lateral sinus trouble over left side. With elimination of all other systemic foci, including plasmodium, renal, pelvis, etc., we feel that X-ray of mastoid should be taken, diagnostic incision of left tympani done and Queckenstedt-Toby spinal fluid test for lateral sinus should be taken. Left sinus should certainly be eliminated.

On Sept. 8, paracentesis of left drum was performed with small amount of suspicious blood-tinged fluid exuding. Spinal tap then done with Queckenstedt's test under gas anesthesia. Normal level of mercury seemed to be 14 m.m., rising as child attempted to vomit. Under better anesthesia it remained at this level. Pressure over left jugular gave no rise in the manometer, while pressure over right jugular caused it to rise rapidly to 22 m.m. mercury. At this point, since the lungs were clear, a faint mitral murmur heard and abdomen negative, it was decided that in view of the findings and absence of other foci that the mastoid should be opened and sinus explored and if thrombosis found, this could account for the condition.

Sept. 9: Patient operated by Dr. Babbit, Dr. Campbell and the writer under gas and ether anesthesia. Usual mastoid incision made behind left ear. Mastoid exposed and curetted—bone rather dense

and bled freely. Upon opening the antrum and probing, pus was seen to exude from middle ear. Lateral sinus then exposed and found to be a yellowish-white in color, with much resistance to pressure. No pulsation and no blood found on aspirating with needle, sinus then opened and thrombosis found. Thrombus cleaned out with probe put in toward torcula until free bleeding occurred and probe passed down toward jugular bulb, but no bleeding was seen. Sinus wound packed with gauze. External jugular vein then ligated, skin closed with silkworm gut and rubber drain. Four silkworm gut sutures to mastoid wound.

Sept. 9: Patient seemed to have stood operation very well. Was not shocked. Pulse rate improved steadily, though intermittent and irregular at times.

Sept. 10: Temperature dropped and was normal today. General condition fair. Pulse of fair volume, but still irregular and intermittent at times. Color poor, with sweating at times.

Sept. 11: Patient slept well last night and temperature remained down, but general condition remains about the same. 4 P. M.: Redressed, sutures removed from mastoid wound, packing loosened and neck wound dressed.

Sept. 11: Patient seemed to be upset by dressing and vomited some curdled milk. Temperature rose again to 105° and patient had a chill. Color very poor and pulse irregular.

Sept. 12: Temperature dropped during night, but up again during afternoon. Patient very constipated, vomited again in afternoon. Milk reduced in diet. General condition about the same. 5 P. M.: Gave 200 c.c. citrated blood, with 100 c.c. saline intravenously; no reaction. Wound redressed and one piece of gauze removed from mastoid wound. Little drainage.

Sept. 13: Patient complained of some headache today and has muscular twitchings. Not confined to any particular group. General condition good. Pulse more irregular and intermittent and respirations irregular. Temperature up to 104.4°. Given 180 c.c. citrated blood and 75 c.c. saline. Redressed and one piece of gauze removed from mastoid wound, and rubber dam from neck. Neck puffy and swollen, but not tender.

Sept. 14: Patient seems definitely better today. Much brighter. Temperature ranging at steadier level, 102° to 104°. Ten c.c. Pregl's iodin given intravenously. Taking nourishment better and no nausea or vomiting. Given 175 c.c. citrated blood and 100 c.c. saline intravenously. No reaction. Blood culture taken. Wound redressed and

all gauze removed from neck. Small amount of pus expressed from lower angle of neck wound.

Sept. 15: Patient gained rapidly yesterday, becoming brighter and stronger. Color improved and today condition seems much brighter. Asking for food. Temperature dropping today. Pulse regular and of good volume. Redressed—packing removed, wound cleansed and packing replaced. Probe into neck wound and small amount of pus expressed.

Sept. 16: Temperature dropped steadily—99.3° today. Patient feeling very well. Pulse still intermittent. Bowels constipated—enema necessary daily. Redressed and gauze packing changed.

Sept. 17: Maximum temperature today, 100.2° at 9 P. M. Normal since. Eating well, resting well. Redressed.

Sept. 18: Condition excellent, redressed, gauze packing changed. Fair amount of pulpy pus expressed from neck wound. Good bowel movement following C. C. pill.

Sept. 19: Patient complained of frontal headache from time to time today and had rise in temperature this P. M. to 103.3°. Pulse and respiration good. Ten c.c. Pregl's iodin given intravenously.

Sept. 20: Patient had slight headache again this morning, but has felt very good and slept at short intervals the rest of the day. Temperature rose again this afternoon to 103°. Redressed; wound looking excellent.

Sept. 21: Patient fair; continues to have slight headache at times. Given 200 c.c. citrated blood and 100 c.c. saline intravenously. Again had temperature rise to 104°.

Sept. 22: Headache in early A. M. and some chilliness. Had rise in temperature to 103° this A. M., dropping during the day. Vomited supper this P. M. Wound redressed and neck healed, so dressing left off. Otherwise condition remains the same.

Sept. 23: Patient had good day today. Temperature rose to 103.3°. No complaints. Seen by Dr. Bacon, who finds no focus for temperature.

Sept. 24: Patient vomited twice today. Complains of shooting pain in right ear last night. Examination of ear and transillumination of sinuses negative. Temperature went up twice in A. M. and P. M. to 103°. Given 10 c.c. Pregl's iodin intravenously. Wound redressed.

Sept. 25: Sent to X-ray and sinuses examined. Patient somewhat more drowsy today and slept much of day. Nauseated and vomited

three times. Diet reduced to liquids. Patient has tendency to keeping head retracted, but not definitely rigid. No Kernig; did complain of some dizziness in P. M.

Sept. 26: Patient looks markedly changed today. Very dopey; to point of stupor at times. Eyes wide open and staring. Head retracted and neck rigid. Legs kept fixed and definite Kernig sign. Complains of dizziness when head is turned. No pain otherwise. 9:30 A. M: Blood pressure, 130-90; pulse good, volume good; temperature, 102°; pulse, 120; respiration, 32. Blood count taken. Spinal puncture done and about 8 c.c. turbid fluid, with flocculent precipitat, removed. Pressure, 6 m.m.; cell count, 65, with smear loaded with Gram positive streptococci. 1:30 P. M.: Seen by Dr. Gardner. Patient somewhat drowsy; co-operation well on examination and answers questions well. Pupils are large, equal and react sluggishly to light. Examination of fundus reveal what appears to be an optic neuritis and not a true choked disc. No engorgement of retinal vessel. No cerebellar signs and no other localizing neurological signs. No nystagmus, asteriognosis, hemianopsia or paralysis of any sort. No loss of sense of position. Biceps reflex slightly diminished, but equal. Left patella reflex very weak, right moderately diminished. Achilles reflex equal, no Babinski, no colonus. With these findings it is difficult to see how child could have a brain abscess, especially in view of low spinal fluid pressure. He has a marked bilateral Kernig and stiffness of the neck. It would seem logical, therefore, to suppose that the child has had a localized meningitis for some time and that this has spread, to become generalized meningitis within past 24 hours. Whether the original focus is above or below the tentorium cannot be decided. Advise liberal use of antistreptococcic serum intravenously and, if well borne, would advise some intraspinally. Lumbar puncture to be performed after intravenous injection of serum and as much fluid withdrawn as will flow out from needle. 3 P. M.: Antistreptococcic serum given intravenously, 30 c.c.; skin test negative to sensitivity. At same time spinal puncture and 25 c.c. of fluid removed. Fluid at 10 m.m. pressure and more turbid than A. M. specimen; substantiated by increase in cell amount to 3,000. No reaction following injection of serum. 8 P. M.: Patient appears somewhat brighter; not so drowsy and eyes not quite so staring. Spinal puncture done and 28 c.c. turbid fluid withdrawn and 10 c.c. antistreptococcic serum injected by gravity intraspinally. Forty c.c. spinal fluid removed. Patient more jumpy and stiffer. Thirty c.c. antistreptococcic serum given intraspinally and 50 c.c. intravenously.

Sept. 27: Patient stuporous this A. M.; more rigid, and pulse rate up to 140, and weaker. Condition very poor. 8 A. M.: Spinal tap; fluid cloudy; 50 c.c. fluid removed. Thirty c.c. antistreptococcic serum given, followed by 70 c.c. intravenously, following which pulse improved and patient became more quiet. 10 A. M.: 185 c.c. citrated blood and 85 c.c. saline intravenously; condition poor. 1 P. M.: Spinal tap; 30 c.c. very thick fluid under pressure removed, and 20 c.c. antistreptococcic serum given, followed by 80 c.c. intravenously. Patient's condition gradually worse. Serum seems to have no effect, and the laboratory failed to agglutinate organisms cultured from the patient, so use is discontinued. 4 P. M.: Patient rapidly growing worse; filling up with mucus and moaning continually; pulse very weak. 4:45 P. M.: Patient died.

Anatomical Diagnosis: Diffuse meningitis. Congestion of spleen and lungs.

External Description: Body is that of a well developed male child, of about 12 years. Over left mastoid area there is an opening in the bone, which is filled with packing. Incision on left neck along border of sternomastoid muscle, about 2 inches long, which is closed by sutures. Both antecubital fossa show small hematomas, with old puncture wounds. The eyes, ears and nose and mouth also are grossly negative. Abdomen slightly distended.

Internal Description: Body opened in usual manner.

Heart: Pericardium and pericardial fluid normal. No endocardial lesions. Heart muscle somewhat pale.

Lungs: Both lungs show passive congestion. No adhesions or apical lesions. Upon the surface of both lungs are scattered a few very shot-like nodules, which are dark gray in color and the size of fine birdshot. These nodules are not surrounded by an inflammatory reaction.

Liver: Shows some cloudy swelling.

Spleen: Enlarged, soft and friable. Dark purple in color; follicles prominent.

Pancreas: No gross pathology.

Kidneys: Show only some cloudy swelling.

Abdomen: Intestine and stomach normal.

Brain: Cranium removed in usual manner. Dura intact. Moderate amount of edema. At base of brain a small amount of thin, seropurulent material was seen. Optic and other cranial nerves were very friable and tore easily. No thrombosis of any sinuses. The opened mastoid was perfectly clean and clear, also the right mastoid. No pus in either internal ear.

Laboratory Findings: Urine: Showed a faint trace of albumin throughout the whole course of illness. S. G. ranging from 1,012 to 1,027. Occasional hyaline casts and a few urates.

Blood Count:	9/6/28	9/26/28	9/27/28
Hemo.	75%		
R. B. C.	4,000,000	3,880,000	
W. B. C.	11,800	32,300	43,100
Poly. Neutr.	65	76	86%
Lymph.	25	17	5
L. Mono.	9	7	6
Trans.	1		2
Eosin.			1

X-ray: Mastoids, Sept. 8, 1928: Slight haziness of left mastoid, indicating congestion. No cellular destruction. Right mastoid is clear. *Sinuses,* Sept. 25: Frontals, ethmoids, maxillary and sphenoid sinuses are clear. *Mastoids:* Right mastoid practically clear. Left mastoid cells show a cloudy appearance; five plates.

Blood Culture: Sept. 6, 1928, negative; Sept. 14, negative.

Spinal Fluid Cell Count: Sept. 8, 1928: Cell count, 1; globulin, very faint trace; sugar, very faint trace. Sept. 26: Cell count, 65; globulin, positive; sugar, negative; smear, Gram positive streptococci, polymorphonuclear predominating, few small lymphocytes. Sept. 26: 3,220 cells; smear loaded with Gram positive streptococci. Sept. 27: Cell count, 1,872; smear, many Gram positive streptococci, polymorphonuclear predominating, some large and small lymphocytes. Sept. 27: Cell count, 3,351; smear, Gram positive streptococci, polymorphonuclear predominating, large and small lymphocytes.

Culture of Spinal Fluid, Sept. 21, 1928: Hemolytic streptococci.

Culture from Mastoid, Sept. 9, 1928: Streptococci, hemolytic.

Summary of Treatment: Ice cap to head. Mag. sulphate. Pot. citrate grs. X. Proctocylsis with whiskey. Digalen M X, q 4 hours, cut to M W. 5 c.c. whiskey t.i.d. Pregl's solution. Antistreptococcic serum. Transfusions.

Diet: Liquid diet on admission; as condition improved, was put on semisolid diet and later returned to liquids.

Pregl's Iodin Solution: 10 c.c. intravenously, Sept. 14, 1928; 10 c.c. intravenously, Sept. 19; 10 c.c. intravenously, Sept. 24.

Antistreptococcic Serum: Sept. 26, 1928: Intravenously, 25 c.c., 2:30 P. M.; intraspinally, 10 c.c., 8 P. M.; intravenously, 40 c.c., 8. P. M. Sept. 27: Intraspinally, 20 c.c., 1:30 A. M.; intravenously, 50 c.c. (50 per cent), 1: 30 A. M.; intraspinally, 20 c.c., 1:30 P. M.; intravenously, 80 c.c., 1:30 P. M.

Transfusions: Sept. 12, 1928: 200 c.c. citrated blood, 100 c.c. saline sol. Sept. 13: 180 c.c. citrated blood, 75 c.c. saline sol. Sept. 14: 175 c.c. citrated blood, 100 c.c. saline sol. Sept. 21: 200 c.c. citrated blood, 100 c.c. saline sol. Sept. 27: 175 c.c. citrated blood, 100 c.c. saline sol.

SUMMARY.

1. Insidious onset, with continued fever as predominating symptom.

2. Question as to time of onset of this illness — did it begin Aug. 1 or Aug. 21?

3. Tonsillar infection seemed to play an important part.

4. Question as to what swimming had to do with this infection.

5. When was lateral sinus first infected.

6. Absence of symptoms accompanying lateral sinus infections, except the Queckenstedt-Toby test and septic temperature.

7. The reliability of Queckenstedt's test in obscure cases.

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TESTS FOR HEARING.

Dr. B. M. Becker, Brooklyn.

An Accurate, Quantitative Method to Test the Auditory Function:

For this purpose 10 feet of soft rubber tubing of about 4/16-inch diameter and 3/16-inch lumen are required. The tubing is divided into three sections, two of which are 24 inches long and one 72 inches long. They are connected in the following fashion: The shorter ones are slipped over the bifurcation of a Y-shaped piece of hard rubber and the longer one over its stem. Each of the shorter tubes is equipped with an ear tip at its free end and a small metal clamp which is fitted over it.

The rubber tubing is marked off in inches (I use white ink on black tubing). Starting with No. 1 at the insertion of the ear tips, the numbers are continued on each tube to their end, which makes it 24 equal divisions, 1 inch apart. The numbers are then continued on the longer section of the tube, beginning with No. 25 and ending with 96.

This testing device can be used with watch acoumeter and forks for each ear, separately or conjointly.

The ear tips are inserted into the patient's ear and a watch is firmly pressed against any number on the tube so as to partly obliterate its lumen. If patient hears the tick distinctly, we proceed down the tube one or more inches at a time until we reach a number where he no longer perceives its sound. We mark the number on our chart and proceed to test the other ear in the same manner. Care must be exercised in clamping the opposite tube from the one used, so that no sound reaches the opposite ear from the one tested, and when starting to test the other ear to open the clamp on side to be tested and clamp the opposite tube.

After we have determined the limit of perception in inches for each ear we express the result either as an integral number, if both ears are equifunctional, or in the form of a fraction (if the ears are not equifunctional), the numerator of which represents the limit of perception for worse ear and the denominator the limit of perception for the better ear. Thus, if it is found that the left ear hears the tick at 23 inches and the right at 40 inches, we express the result as 23L/40R, but if both ears perceive the tick at the same number, say 30 or 40 inches, it is sufficient to mark that one number for both ears.

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Now we have something definite and precise to enter in our records for the sake of comparison as patient returns for treatment. Thus, if after one or more treatments we test the hearing in the same manner and find the fraction given above changed to 34L/40R, we know that the hearing for watch has improved in the left ear by 11 inches, whereas it remains stationary for the right ear; or if fraction is changed to 37L/45R, we know there is improvement in both ears, but to an unequal degree. Thus, by marking this on our record we have an exact quantitative index of change or stationary condition.

If examiner enjoys normal hearing, he may mark off on tube the distance of limitation for himself, and use this as standard of comparison, or testing a number of individuals with normal hearing, he will arrive at an average as criterion against which to check patient's hearing. This method is equally applicable with the acoumeter in cases of advanced deafness. The tube is simply pressed against the upper or lower concavity in the frame of the instrument at a given number, and moved up or down according to the perception or lack of perception of the sound produced by its operation. The same precautions must be exercised in the use of the other instruments with the tube, as in case of the watch. Since the forks, in contradistinction to the watch and acoumeter, lend themselves to both distance and duration tests, the method described becomes more intricate, yet more interesting, in that it suggests a subject for further investigation and elaboration.

In testing the hearing of one ear as compared with the other, we proceed as in the case with the other instruments. Gently tap fork or compress its tines so as to produce a mild vibration and set its stem against any number on tube—the opposite tube being clamped. When the sound is no longer perceived by patient, unclamp opposite tube and set stem of fork on its corresponding number. If both ears perceive sound for same length of time at the corresponding numbers on each tube, we record them as equifunctional with reference to the forks. If, on the other hand, one ear still perceives the sound after its mate ceased hearing it, we time the difference in seconds and chart it. Next, we test again, altering the distance of fork for the two ears. Either we move fork down the tube for the better ear, or move it up the tube for the worse ear, until we get a correspondence of time for the perception of the sound, for the two ears. We note the difference in inches and check it against the difference in seconds in our first step, and we have an interesting comparison.

To illustrate: The vibrating fork is set at 20 inches from right ear. After the sound dies down, the fork is set on opposite tube, 20 inches from left ear, and is perceived 5 seconds longer. Next we move the fork up on the right tube or down on the left tube until the sound is heard equally long in both ears. Let 3 inches be the distance we have to move the fork: Then we may state that the right ear lags behind the left either in 5 seconds or in 3 inches. (The above represents what the author found in his own case, using a 128 and a 256 fork.)

In comparing fork time, i. e., the time patient will hear fork as checked against our own hearing according to the tube method, we use one of the shorter branches of the same device. After detaching it from the connector, an ear tip is put in its lower end, and the tips are inserted, one into the patient's ear and one into the examiner's ear, respectively. The stem of a vibrating fork is pressed against the tube exactly in its middle. The sound is carried equally to the examiner and the patient. When the patient no longer hears the sound he informs the examiner by a signal, so that the latter can note on his watch the number of seconds the patient is behind in his hearing. Or he may move the fork toward the patient and, finding the distance in inches on tube where hearing time becomes equalized for both himself and patient, mark the patient's subnormality in inches.

Transvibration: A Combined Objective and Subjective Method for Differential Diagnosis—Preliminary Report:

Politzer states (The Diseases of the Ear, 6th ed.): The vibrations of a body brought in contact with the cranial bones are distributed to all parts of the head, and thus reach the internal ear. These vibrations reach the labyrinth in two ways: *1.* By direct conduction through the solid bones to the labyrinth; *2.* By transmission from the cranial bones to the membrana tympani and ossicles, and from these to the labyrinth (cranis-tympanic conduction).

It is on the second part of the statement that this test depends. When the drum membrane vibrates by its excursions it sets up a disturbance in the air, not only in the tympanum but also in the external auditory canal. If there were, so to speak, a sensitive auditory apparatus external to the drum, the vibrations generated in the tympanic membrane either by bone or air conduction would be equally perceived by it. This is precisely what takes place, roughly speaking, in this test, where the examiner supplies the mechanism.

Two tubes, each 2 feet in length and of equal calibre, at each end of which is inserted an ear tip (of same size and bore) are used for this test. The examiner sits facing the patient and the ear tip of each tube is inserted, one into the patient's ear and one into the examiner's ear.

A tuning fork (32-256), preferably of the two higher denominations, is set vibrating rather forcefully and placed in midline on patient's head. Now, one of two things will be observed by the examiner, in so far as the transmission of the sound from the patient to himself is concerned. *1.* Either the sound will be so pronounced on one side that he will be able to lateralize it with certainty each time the test is repeated. This is the positive (+) phase. *2.* Or the second will reach examiner's ears with equal intensity. This constitutes the negative (—) phase.

In a number of patients examined by this method the following results were obtained: *1.* In cases where one ear is normal and the other affected by a conductive lesion, the sound is transmitted with greater intensity by normal ear. *2.* In conductive lesions of both ears, where the lesion predominates in one ear, the sound will be perceived by examiner with greater intensity on side which is less affected.

While it would be premature and therefore unjustifiable to draw definite conclusions from the phenomenon observed, yet tentatively we may assume from the positive result (provided we can rule out mixed affection) that the ear which transmits the sound more distinctly is either normal or has a conductive lesion of less intensity than its mate.

The subjective component of this test consists in reversing the process. The examiner places fork on his own head and patient informs him where he hears sound better. The subjective component of this test, together with the other recognized means of diagnosis at our disposal, will furnish data to check up on the objective phase of this method.

Care must be exercised in this test to exclude obstructions or malformations of patient's external meatus, and to be sure that examiner has equifunctional hearing. If in doubt about this latter point, it is well for examiner to interchange the tips in his ears by crossing the tubes.

4401 17th Avenue.

MANAGEMENT OF ABSCESS OF THE NASAL SEPTUM.

DR. IRVING WILSON VOORHEES, New York.

Although abscess of the nasal septum is one of the commonest of intranasal conditions and is easily diagnosed, it is not treated with uniform success. We have all seen cases that have run on for from four to six weeks, or until no further destruction of the septum could take place. The result in such conditions is retraction of the flaps owing to scar tissue formation, and a drawing in of the lower third of the external nose to produce a "saddleback" effect. Not infrequently this deformity has given rise to litigation in the courts, and the defect which is so very obvious to a jury makes the defendant's chances of a favorable verdict extremely slight.

The common causes of septal abscess are trauma, such as a blow from a fist, infected hematoma and submucous resection of the septal cartilage for a deviation from normal perpendicularity. Since the submucous operation has become so common, one sees more abscesses following operative interference than from any other single cause. No matter how careful one may be in his operative technique, there is no such thing as complete sterilization of the septal field; therefore, the surprising thing is that we do not have more rather than less infections between the flaps. After a submucous there is in some cases a great tendency to oozing of blood and serum, so that no matter how well the nose is packed with the design of producing complete coaptation and adhesion of the flaps and a straight septum as a final result, the ballooning of the interflap space persists, so that the nose is fully obstructed and mouth-breathing must go on, to the great distress of the patient.

Several years ago a woman, about 30 years of age, came to the Manhattan Eye, Ear and Throat Hospital and was assigned to me for the relief of nasal obstruction. She said that she had been under treatment by a famous or notorious advertising beauty specialist, who injected paraffin into a depression at the tip in order to raise it. The paraffin had spread into the adjacent skin of the nose and face, and into the septum, setting up there an abscess which destroyed the nasal support and making the original deformity much worse than it had originally been. She was treated in the way about to be described and eventually got a good result from a rib transplant. I was brought

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into court as an "expert" witness, but simply related what has been told here. She won a verdict of $3,500, but had some difficulty in collecting it I believe.

Another patient, a law student, had a successful resection of the septum performed and was about ready for dismissal after some 10 days of after-care, when he got into a fist fight and "stopped a fast one", as he expressed it. An abscess between the flaps developed. he was treated by another rhinologist in a different hospital and eventually recovered. But when Surgeon A tried to collect a fee for his operative work, he was confronted by a counter-suit for having taken out too much support, and not responding to a call for his services, a distance of some 10 miles, when the young Blackstone got into trouble. The patient was about 20 years of age, and in the eyes of the law, an "infant". The surgeon had not obtained either permission or authorization from the boy's parents before proceeding with his operation; therefore, he faced a technical charge of "assault" and was lucky to get out of the trying situation without paying damages, let alone collecting his just fee. None of us, or better, perhaps, few of us are equal to matching wits with such unscrupulous persons, but we ought to know that they exist, nevertheless.

My method of handling these abscess cases is, I think, original and I have used it for some 15 years without reporting it to the medical societies or "writing it up".

If a recent submucous incision is present, it is reopened with a sharp knife and the fluid contents of the cavity are removed by suction. Local anesthesia is used, but as the procedure causes very little pain, one need not be meticulous about waiting for deep absorption. In fact, the tissues are so waterlogged that it is not possible to get great penetration into the cells. Following the suction, a flap speculum is put in place and the cavity washed out with normal saline. The area is thoroughly dried with gauze or cotton. One then makes a small cotton swab and dips it in pure phenol, making sure that there is no excess which can run down over the skin. The cavity is swabbed lightly, but thoroughly, and a second swab dipped in 95 per cent alcohol is brushed over the surface in the same manner, so-called "dehydrating". Finally the flaps are held together by the flap speculum, introduced on the *outside* of them instead of in between, thus securing coaptation, and a Bernay splint or Simpson nasal tampon is put into each nostril in order that gentle pressure shall be maintained. Two or more of these tampons may be required, according to conditions present. They are left in place for 12 or 24 hours, never more than 24, and when removed the condition is

practically relieved and nothing further is required save occasional observation to see that all is going well.

The method which was taught in my student days was incision and introduction of various drains, such as rubber tissue dam, small tube rubber, etc. While we used to get them well in time, I think the period of convalescence was too long as compared with the simple procedure here outlined.

114 East 54th Street.

EXOPHTHALMOS DUE TO CHRONIC ABSCESS OF ORBIT AND CHRONIC FRONTAL SINUSITIS.*

DR. C. G. COAKLEY, New York.

Mrs. C. E. B., age 73 years, was referred on Sept. 24, 1928, by Dr. J. W. White. The patient had had exophthalmos for three months, with some conjunctival inflammation. She had slight diplopia, but no headaches or sharp pain. She had also been seen by Dr. Knapp, for whom radiographs were taken, and reported a dense mass, possibly osteoma, in outer part of right orbit. Owing to nasal obstruction and some discharge, it was thought wise to have a rhinological examination. In my absence, Dr. Babcock saw the patient in consultation with Dr. White. The right naris contained much pus, and the middle and inferior turbinates were greatly swollen and edematous, the middle turbinate appearing cystic. There was marked exophthalmos and a hard mass was felt just below the outer half of the orbital arch; it was not painful. On transillumination, the right frontal and maxillary sinuses were very dark; the left, good.

The right antrum was irrigated through the nasal wall below the inferior turbinate and much foul-smelling pus was removed. Smears and cultures were sent to Dr. Sondern, who reported a purulent secretion with a growth of pneumococcus and staphylococcus aureus.

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*Read before the New York Academy of Medicine, Section on Laryngology and Rhinology, March 27, 1929.

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At this time the diagnosis lay between a tumor of the orbit complicated by paranasal infection, and a paranasal sinus suppuration complicated by an orbital abscess. Suspecting the latter, Dr. Babcock, on Sept. 27, made a large opening in the right nasoantral wall for drainage of the maxillary sinus. On Oct. 1, Dr. Babcock amputated the anterior end of the right middle turbinate and opened some neighboring ethmoid cells to facilitate the irrigation of the right frontal sinus. The parts were so vascular that he was suspicious of a malignant neoplasm and sent the specimen to the Presbyterian Hospital for microscopical examination. They later reported no evidence of malignancy.

I saw the case first on Oct. 4, 1928, and my impression was that of an orbital abscess complicating a chronic sinusitis. On Oct. 8, a further intranasal exenteration of the anterior and middle ethmoids was done by me, hoping to reach the orbital abscess and facilitate treatment for the right frontal sinus. As a result of this we were not able to reach the orbital abscess but were able to pass a cannula into the right frontal sinus and evacuate a quantity of foul pus. The examination of the nose with the nasopharyngoscope and probe showed that the sphenoid on that side was not involved in the infective process.

On Oct. 15, the nasopharyngoscope was placed in the right naris so as to view the lateral wall, and pressure backward was made on the protruding right globe. This was followed by seeing some thin pus appear through the granulation in the ethmoid area. As a result, we felt we were surely dealing with an orbital abscess, but still could not find the fistulous tract into the orbit. It also seemed as if the mass in the orbit was less tense. This was admitted by the ophthalmologists, and for the first time they were willing to concede the possibility of an orbital abscess, though not convinced that there might not also be a tumor, probably malignant. In the meantime the right maxillary sinus had ceased discharging.

On Oct. 25, at the Presbyterian Hospital, Dr. Bennett administered the anesthetic and, assisted by Dr. Babcock and Dr. Brighton, operation was performed. It was expected that an external incision from the supraorbital notch inward and downward would enable us to exenterate all the ethmoidal cells, expose the inner side of the orbit, and enable us to find the communication with the abscess and evacuate it. After doing this, we were no nearer the end-result, but only saw through the floor of the frontal that we had a very badly abscessed frontal sinus. We then made a horizontal incision through the eyebrow, removed the anterior wall of the sinus and thoroughly

cleaned it out. There was a long prolongation toward the outer angular process, but no communication with the orbit. On removing the roof of the orbit at its outer portion, we noted that the exposed tissue was a dark purple instead of the normal light yellow. After a longer exposure, a careful incision into this region was followed by a flow of thick, foul-smelling pus from a cavity the size of an English walnut and extending back to the apex of the orbit. The cavity was surrounded by a dense fibrous membrane.

Microscopical examination showed no evidence of malignancy. The entire orbital arch was removed and the cavity was made to communicate with the nose through the ethmoid area.

Both wounds were closed and drains brought from the orbit out at the naris. These were removed in 24 hours. Primary union was obtained in the frontal and ethmoid incisions. By Nov. 9, the crusting and discharge in the nose had practically ceased.

On Nov. 13, a slight swelling appeared at the inner canthus, which gradually increased and spread to the upper lid—evident infection. On Nov. 18, slight fluctuation was detected in the upper lid, which was incised and pus evacuated and a probe was passed 1 inch back toward the apex of the orbit. This sinus continued to drain slightly, until on Dec. 29 the sinus was injected under pressure with bismuth paste, and the patient was radiographed. The plates showed a cavity still at the apex of the orbit. This cavity has alternately remained active and quieted down after evacuation until the present time. To accomplish a cure, it will be necessary to again expose the cavity and curette the thick wall more thoroughly.

20 East 53rd Street.

IRRIGATION OF ANTRUM IN INFANTS AND CHILDREN THROUGH NATURAL ORIFICE.*

DR. IRVING B. GOLDMAN, New York.

That disease of the maxillary antrum plays an important role in the diseases of childhood is recognized and undisputed. Clinical and post-mortem studies within the past decade have shown the large amount of antrum infections in infants and children. Other sinuses, indeed, are also involved but the maxillary antrum is the cavity where diseased conditions have been observed most often.

Despite this knowledge, too large a proportion of cases go undiagnosed and too small a proportion receive proper and adequate treatment. Our purpose in this presentation is to describe the procedure which we have been using successfully in the treatment of maxillary sinusitis.

The puncture of antra in the first years of childhood is difficult and hazardous. It is hazardous because the bone below the antrum may be easily penetrated, or the anterior wall of the antrum may be perforated. The anatomical outlay makes puncture difficult. The meatus is low and narrow, the mucous membrane of the inferior turbinate is rather thick, and often the lower border of this is in contact with the floor of the nasal cavity and entirely blocks the lower meatus.

We have eliminated the necessity of puncture of the antrum through the inferior meatus in a vast majority of cases by irrigation through the natural orifice. Although the antrum is small, the natural orifice is relatively large. In the infant cadaver the hiatus semilunaris can be seen to be a rather broad gutter, at the posterior extremity of which is found the ostium of the antrum. For the purpose of entering this opening we have been using miniatures of the natural orifice cannulae, devised by Dr. Yankauer for adults. The cannula is 3 inches (7.6 c.m.) long and is of a small bore. At the distal end it is curved at right angles to the shaft. The curved arm is of three sizes, ¼-inch, ⅜-inch and ½-inch. These are intended for the children of various ages, the older the child, the larger the curved arm used. There are right and left cannulae.

Technique: The nose of the infant is sprayed with epinephrin hydrochlorid in order to shrink the mucous membrane of the intra-

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nasal structures. Sometimes, in order to get a more patent middle meatus an applicator with the solution is placed in the middle meatus. The cannula is then introduced into the nose so that the curved arm passes along the upper surface of the inferior turbinate with the tip facing the lateral nasal wall. As the hiatus semilunaris is approached, the tip of the curved arm is rotated outward and downward and is directed into the natural orifice. The flexibility of the soft structures of the middle meatus guides the canula towards the orifice when the hiatus semilunaris is reached. As the orifice is entered a peculiar sensation will be imparted to the fingers of the manipulator. Older children must have nasal anesthesia before they will permit manipulation. We spray the nose with a 4 per cent cocain solution containing adrenalin, wait a few minutes for the drug to become effective, and then apply a swab of cocain to the middle meatus. When the anesthesia is complete and we are certain that there will be no pain, the cannula is inserted in the manner described. Success in this procedure requires the knowledge of the location of the ostium and the employment of the cannula as one would a probe.

The following conveys an idea of various ages of infants where antra were entered through the natural orifice: 6, 10 and 14 weeks, 3, 5, 6, 11, 14, 21 and 24 months, and older children.

Catheterization of the antrum through the natural orifices has given splendid and very interesting results, which will be dwelt upon in a subsequent paper. We advocate this method because it is feasible and can be easily carried out. It appeals to the parents of the young, who do not consider it an operation and are willing to have the children subjected to this procedure.

121 East 60th Street.

IRRIGATION OF ANTRUM.*

DR. SIDNEY YANKAUER, New York.

It is not as difficult to pass the antrum cannula through the natural orifice of the antrum in a child as in an adult because the natural orifice of the antrum in a child is comparatively larger. In adults we are able to enter the natural orifice in only 50 per cent of the cases. In cases which cannot be cannulized, it is necessary to puncture. There are two methods of puncturing the antrum—through the lower meatus and through the middle meatus. Where an attempt has been made to pass the natural orifice cannula, the middle meatus is already cocainized and it is therefore preferable to puncture in the middle meatus. Cannulae for puncturing the antrum in the middle meatus have been devised by von Eicken, Abraham and Killian. The best known of these is that of Killian, which consists of a cannula bent at right angles. This instrument has proven to be dangerous because the floor of the orbit sometimes sags down to a considerable extent, so that it comes to lie almost parallel with the outer nasal wall in a position corresponding to the natural antrum orifice, so that the orbit can easily be injured if a cannula is forced horizontally through the outer nasal wall in this region.

Experience with the natural orifice cannula, as this instrument has been shaped by the writer, has shown that when it is passed through the natural orifice it is impossible to come in contact with the orbit, for no matter how near the orbit may come to the outer nasal wall, it is necessary to rotate the cannula in order to introduce it through the natural orifice. This rotation immediately brings the point to a downward position away from the orbital floor. Therefore, for the purpose of puncturing the antrum in cases where the natural orifice cannula cannot be passed, I have constructed a trocar shaped exactly like the natural orifice cannula, but sharp at the end, and this instrument we have tried out for this purpose. The result was not merely surprising; it was astounding.

The location which is penetrated by the instrument is an area in which the bone is normally dehiscent and nothing but mucous membrane closes this dehiscent area. It is not necessary to pass through

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bone in order to enter the antrum. In fact, the mucous membrane closing the dehiscent area is so delicate that the sharp point of the instrument passes through it without giving any sensation whatever, either to the patient or to the surgeon. In many cases it is not possible to sense the moment when the instrument passes through the antrum wall. It is an entirely different procedure from puncturing the antrum in the lower meatus, for there is a complete absence of the sound of crushing bone—there is no sudden, jerky movement —there is no force necessary. In fact, the two procedures cannot be compared; they can only be contrasted.

I have been using this instrument for about nine months and during the recent grippe epidemic there were so many cases of maxillary sinusitis that I am not exaggerating when I state that we have used this cannula hundreds of times. Puncture of the antrum is no longer a matter of fear or dread for the patients and many times we have carried out the procedure without any realization on the part of the patient of what was being done.

The instrument is manufactured by The George P. Pilling and Son Co., Philadelphia, Pa.

121 East 60th Street.

RECURRENT POLYPI OF MAXILLARY SINUS IN A BOY OF EIGHT YEARS.*

DR. J. W. BABCOCK, New York.

The comparative rarity of nasal polypi in children made me consider this case of sufficient interest to report. Master T. S., white, age 8 years, had a history of leftsided nasal obstruction of two years' duration, when first seen by me on Nov. 27, 1928. He had had polypi removed from the left naris under general anesthesia twice during the previous six months, and a Caldwell-Luc operation had been advised. With some difficulty, I persuaded him to let me remove two large polypi under local anesthesia, and thought, since he admitted that it did not hurt, that further general anesthesia would be unnecessary. In that I was mistaken, for he absolutely balked at two subsequent visits, and on Dec. 7, 1928, under general anesthesia I removed a large number of polyps from his left naris and made a large intranasal opening into his left maxillary sinus. He looked well until Dec. 24, when polypi appeared at the opening into the antrum, preventing the irrigation which he had been receiving at the hands of Dr. Michaelis and Dr. Harrison.

On Feb. 18, 1929, after some training, the boy submitted nicely to having the polypi present in the antrum removed intranasally under local anesthesia by Coakley curettes and forceps. Since then no polypi have been observed, although some mucopus still accumulates in the antrum, as shown by irrigations.

Pathological examination on two occasions showed simple nasal polyps. Radiographs showed cloudiness of the left maxillary sinus only.

The points of interest to me in this case, aside from its comparative rarity, were that so much could be accomplished intranasally and that, with training, a very difficult child could learn to co-operate in treatments. He even served as an example to another frightened child recently, after having appeared to me for a time as the worst patient I had ever attempted to treat.

20 East 53rd Street.

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LYMPHOSARCOMA OF THE TONSIL.*

DR. GEORGE V. BROWNE, Long Island City.

The patient, a man age 39 years, was admitted to the hospital for the first time in September, 1927, for an undiagnosed condition of the testicle, which had been more or less swollen for two years. His family history was negative, as was the blood Wassermann test. He had had an appendectomy performed in 1910.

On Oct. 5, an orchidectomy was performed, and a laboratory diagnosis was made of carcinoma of testicle. His convalescence was uneventful, and he was discharged in December after being in the hospital for two months.

In June, 1928, he returned, complaining of loss of weight, constipation, gastric symptoms and general asthenia. He was referred to the nose and throat department, and examination showed enlargement of the left tonsil, with an extension of the lower pole, which had the appearance of a new growth. He was then referred to the medical department to rule out any gastrointestinal involvements. A gastrointestinal series was taken, but nothing definite was made out.

Tonsillectomy under local anesthesia was performed on July 26. There was very little bleeding, and the tonsils and capsules came out intact. The patient was put to bed, and that night vomited about 500 c.c. of clotted blood. He had upper abdominal pains and was very rigid; his pulse was weak; the heart was rapid and sounds very poor. Morphin was given, and a diagnosis was made of ruptured viscera due to the metastatic condition. He was too weak to endure an exploratory abdominal operation. More morphin was given, and it was felt that he would expire very shortly. However, next morning he seemed to be in a much better condition, and was transferred to the medical ward. He remained there 12 days and was then discharged.

Microscopic Diagnosis: Lymphosarcoma of the tonsil.

196 Van Alss Avenue.

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REPORT ON CASE OF COMPOSITE TUMOR OF TONSIL.*

DR. GEORGE V. BROWNE, Long Island City.

The patient, a colored woman age 33 years, was admitted to the hospital, complaining of trouble in the throat with which she had suffered for 20 years. During the past few years it had been growing worse and patient had difficulty in swallowing. A year before she had been in the hospital for lues.

Examination revealed no palpable glands in the neck. In the throat was a large mass on the left side of the palate, about the size of a lemon, completely hiding view of the other tonsil and uvula. The mass appeared hard and had a round punched-out area. A small section of this tissue was taken for examination and was reported on as follows:

"Gross: The specimen said to have been taken from the right side of the pharynx is a minute mass of firm white tissue measuring 5x4x2 m.m. On one surface it has a brownish central area, which is surrounded by a thick white area, which may be mucous membrane.

"Microscopic: Sections of 37417 shows stratified squamous epithelium lying on a fibrous stroma which contains many dilated vessels immediately beneath the epithelium. There are irregular masses of cells, which are most numerous in the subcutaneous tissue but which are also found in the corium. The cells are fairly uniform in size and have round or ovoid nuclei which contain a moderate amount of chromatin. Their cytoplasm does not stain clearly but contains fine fibrils. The cells frequently form columns two cells wide. No lumen is seen between the columns. Scattered round cells and plasma cells are seen throughout the stroma.

"Diagnosis: Composite tumor of pharynx."

This tumor was removed in the same order as an ordinary tonsillectomy, and the postoperative treatment was the same. Several weeks later the patient returned with the area completely healed, and with no further complaints.

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Laboratory report of specimen after operation: *"Gross:* Specimen is a large, nodular, encapsulated, firm tumor mass, which has an anterior surface which appears to be covered with mucous membrane similar to that usually seen in tonsils. In this area there is an irregular, brownish, caseous surface about 7 m.m. in diameter, which suggests an ulceration. The mass measures 5x3x5 m.m. On section, the surface is extremely fibrous, white, with heavy connective tissue trabeculae, some of which seems to contain cartilage. There are several discolored areas surrounding what appear to be crypts. There are also a few dilated cystic cavities measuring about 2 m.m. in diameter, which are filled with a pale brown, clear fluid. There is a second specimen. On section, it has an extremely firm, fibrous stroma which appears to contain lymphoid tissue.

Microscopic: Sections from the large mass show a portion of the fibrous capsule at one margin. The bulk of the tumor is composed of cells which contain round, or almost round, nuclei lying in a fibrous stroma. The cells are a little larger than a small lymphocyte, with a small amount of cytoplasm, which takes a uniform pink stain. The nuclei do not vary much in size; they take a moderately deep stain of almost uniform intensity. No mitotic figures are seen. There are areas of fibrous tissue in the tumor where these cells are scarce, and several empty spaces presumably representing the cysts seen in the gross. One of these spaces contains a substance and is lined by flattened cells. Sections from the smaller mass show a tonsil with epithelium of varying thickness, debris in the crypt, and a thick capsule which contains lymphocytes.

Diagnosis: Composite tumor of pharynx: chronic tonsillitis.

196 Van Alss Avenue.

AGRANULOCYTIC ANGINA CASE REPORT.*

DR. WILLIAM SPIELBERG, New York.

Agranulocytic angina, agranulocytosis, agranulocytic leukopenia or malignant leukopenia are terms used to designate a condition, disease or symptom complex characterized by a sudden onset of sore throat, severe prostration, high temperature, which rapidly terminates into coma and death.

The two outstanding clinical features of this condition are: *1.* Ulcerated and necrotic areas in the mouth and pharynx; *2.* a very low white blood count, particularly in the number of polymorphonuclear leukocytes (granulocytes), which before death takes place may entirely disappear from the blood picture.

Historical: Agranulocytic angina was first described by Turk in 1907, and by Schultz in 1922. In 1924, Lovett described a case in this country. This was a fatal case of ulcerative stomatitis with extreme leukopenia in which the bacillus pyocyaneus was associated as the probable cause. Since then a large number of cases of agranulocytic angina have been reported in this country.

In 1927, Fred. H. Linticum reported a case of agranulocytic angina where the bacillus pyocyaneus was recovered from the slough in the mouth. This case terminated fatally on the eleventh day with necrosis and sloughing of the gums between the teeth, with exudate and fibrinous bands extending from the gums to the cheeks. Death was preceded by dyspnea, high temperature, involuntary bowel movements and coma.

E. C. Piatt, reporting on the histopathology of agranulocytic angina, describes a Gram-negative bacillus which he thinks belongs to the bacillus pyocyaneus species. A. S. Wieder reports a case where one patient suffered two attacks, the second attack proved fatal. The organism is not mentioned. R. J. Hunter reports a case following fracture of the tibia. H. P. Hul reports a case following extraction of teeth, who developed a gangrenous process of the mouth and nose. This patient had a total white count of 1400, with 3 per cent granular cells (polymorphonuclear leukocytes); the patient recovered. B. pyocyaneus were not found.

Wm. C. Heuper and D. O'Connor report five cases of agranulocytic angina, all terminating fatally. Three were women and two

*From the Otolaryngological Department of Beth Israel Hospital.

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were men. The ages varied from 36 to 79. The authors do not give any positive etiology. They think that the presence of streptococci and other bacteria in the blood is not a primary but a secondary condition, due to an invasion of these bacteria through necrosis in the mouth.

V. K. Hart reports a case of combined Ludwig's angina, agranulocytic angina and septicemia; with a similar case in a sister of this patient. Both terminated fatally. Both had positive blood cultures. The first had streptococci and staphylococci, and the second, Friedlander's pneumobacillus. The author considers both as real cases of septicemia. The first case died within 36 hours of admission and five days from the onset of the first symptom. In the second case, death occurred within three days of onset.

Etiology and Pathology: Not very much is as yet known of the etiology of this disease. It is, however, believed by some to be a virulent primary throat infection with an overwhelming toxic paralysis of the blood-forming organs. Others believe it to be a blood-stream infection with the throat as the portal of entry. Smears and cultures taken from these patients show a variety of organisms; hemolytic and nonhemolytic streptococci, short chain streptococci, encapsulated pneumococci, staphylococci, Vincent's organisms. The bacillus pyocyaneus and Friedlander's pneumobacillus were found in a few cases. It is believed by some that the bacillus pyocyaneus plays an important role in cases of pyocyanic stomatitis and agranulocytic leukopenia. This type is considered by Linticum to belong to the fatal group. He has recovered the bacillus pyocyaneus in pure culture from typical lesions as well as from the internal organs. Broth cultures of pyocyaneus made from the mouth of one of these patients, injected into guinea pigs, showed it to be definitely pathogenic to these animals, and seems to possess the power of either destroying or inhibiting the formation of granular elements in the blood, also having the power to produce a leukopenia. Other organisms if given in sufficiently large doses may also produce a leukopenia but do not prove fatal to the animal.

Pathologic sections made of tissue show a vast number of bacteria and is probably due to a great reduction in the number of polymorphonuclear leukocytes, than the severity of the infection. Autopsies done on some of the cases show no characteristic lesions in the heart, lungs, kidneys, spleen or liver, while in others acute degenerative changes in the heart, liver, kidneys, etc., have been noted, as well as evidence of acute infection. Ulcerative lesions have been found in the stomach, duodenum, ileum, colon, anus, rectum, cervix

and vagina, and on the symphisis, hip and conjunctiva. The tonsils were always involved and in most cases reported, the tongue and gums. One case showed involvement of the larynx. Examination of portions of the membrane from the mouth obtained during the early stages of the disease, revealed a variety of organisms, among which have been found actively motile Gram-negative bacilli which were identified as being bacillus pyocyaneus. They are differentiated from the diphtheria bacillus by the Gram stain, the latter being Gram-positive. The bone shows a distinct decrease in white cells, with few or no granulocytes or myelocytes and many lymphocytes. The erythrocytes are in normal number.

Symptoms and Course: The onset of the disease is sudden, with sore throat, accompanied by high temperature, chill, headache, general malaise and pains throughout the body. Examination of the throat reveals a marked congestion of all mucus surfaces of the throat and mouth, with ulcerations, single or multiple, involving primarily the tonsils. Lesions may also be found on the cheeks, tongue, gums and larynx. As the disease progresses there is formed a dirty, gray, fibrinous exudate upon the tonsils, resembling the coalescent form of follicular tonsillitis, or more frequently the diphtheritic membranous exudate. Similar necrotic processes with this exudate have been found on the skin, in the vagina and intestinal tract. Further general examination does not reveal any characteristic findings except such as might complicate the disease before death ensues. The glandular system is not usually involved. Our case showed a marked involvement of the superficial and deep cervical and subclavicular glands on the right side, which grew to enormous size before death took place. The course of the disease is usually fatal and, according to some authors, always terminates fatally where the bacillus pyocyaneus is found in smears from the throat and in blood cultures. The urine examination shows albumin and casts.

The Blood Findings: The characteristic diagnostic feature of this disease is in the blood picture. The red blood cells, hemoglobin and blood platelets are normal or slightly reduced. The white blood cells are reduced to as low as 500. The polymorphonuclear leukocytes are greatly reduced or absent (agranulocytic). The lymphocytes, although relatively increased, are actually decreased. In our case, only 50 cells were counted in making the differential count, due to the difficulty in finding them. Blood cultures have usually been found negative, but the streptococcus hemolyticus and the bacillus pyocyneus have been found. The coagulation and bleeding time are normal.

Differential Diagnosis: Many cases reported as agranulocytic angina cannot truly be recognized as such. They are really septic infections with agranulocytic symptom-complex and have been observed in such disorders as pernicious anemia, sepsis, kala-azar, noma, leukemia (acute lymphatic), acute lymphadenosis, abdominal lymphogranulomatosis, carcinoma of the bone, poisoning from benzol, salvarsan and X-rays, etc.

Agranulocytic angina has to be differentiated from septicemia with a low leukocyte count; from acute sore throat with cervical adenitis having an increased lymphocytic count, better known as acute lymphadenosis or infectious mononucleosis. From lymphatic leukemia it can be differentiated by its usually higher white count, reduced hemoglobin, red count and low color index. According to some authors we are asked to distinguish between two manifestations of this disease:

1. Agranulocytic angina that may occur with any severe stomatitis, occasionally occurs in men and has no characteristic bacteriology, has a leukopenia with diminution in the granular elements of the blood and do not necessarily terminate fatally. In this group are classed all cases of agranulocytes angina.

2. A group of cases occur in middle-aged women, which in cases so far reported proved fatal, and in which the bacillus pyocyaneus has been recovered in pure culture from the typical lesions or from the internal organs. For this group is suggested the name, "Pyocyanic Stomatitis with Agranulocytic Leukopenia".

Treatment: The therapeutic means employed in the treatment of agranulocytic angina may be divided into local and general. The local treatment is chiefly directed towards the alleviation of the pain and soreness of the throat and mouth. Local applications are employed, such as the arsenical compounds, chiefly salvarsan and neosalvarsan. A 10 per cent solution of neosalvarsan in glycerin freshly prepared is preferred, and is swabbed over ulcerative surfaces. Mouth washes and gargles of antiseptic solutions are given for cleaning the mouth.

For general means, salvarsan or neosalvarsan intravenously have been used. Streptococcus antiserums, blood transfusions and intramusclar injections of blood have been employed. One patient was treated with diphtheria antitoxin. Radiation of the long bones with stimulating doses of X-ray have also been tried.

Report of Case: History: S. O., male, white, Russian, admitted to the Beth Israel Hospital, June 14, 1928, at 1:30 a. m.; died June 15, 1928, at 2:30 p. m. Chief complaint: Sore throat and difficulty

in swallowing. On June 13, 1928, at 2 p. m., I was summoned to the patient's house. I found him lucid, responding to all questions. He complained chiefly of a severe pain in the throat and inability to take even fluids. On attempting to swallow the fluid, regurgitated through the nose.

Present History: Began four days ago with cold. Patient complained of generalized pains, drawing in character. He took to bed, and the next day experienced a soreness of the throat, which rapidly became so severe that he was unable to swallow. About the same time he also developed a painful swelling at the angle of the right jaw and the side of the neck. The day I was called he had a severe chill and his temperature went up to 105° F.

Examination: General appearance was cyanotic and somewhat dyspneic. Lips were swollen, with a profuse flow of saliva from the mouth, somewhat mucoid in character.

Rhinoscopic Examination: Did not reveal any unusual findings.

Examination of Pharynx and Mouth: Revealed the mucous membrane to be of a dark, livid hue. The tonsils submerged and covered with a dark, dirty, gray exudate, which was hard to remove and left a bleeding surface. The tongue was extremely tender, especially at the base. The neck was very tender, mostly at the right side in the submaxillary region and at the angle of the right jaw, where there was a considerable swelling of the lymphatic glands. Submaxillary and post-occipital glands were found swollen and extremely tender. Temperature, 105°; pulse, 120; respiration, 28.

I prescribed for the patient and five hours later I was again summoned to the patient's bedside, to find him in such a critical state that I advised his immediate removal to the hospital.

The following history and physical examination is from the records of the hospital, taken immediately after admission: *Physical:* Male, 60, well built, sallow complexion, Cheyne-Stokes' respiration. *Eyes:* Pupils react to light and accommodation. *Mouth:* Dental curries, pyorrhea, dry tongue with patches of white exudate. Pharynx very red and purplish area on right side. Tonsils are covered by a dark gray exudate. *Neck:* Tender, and swelling at angle of right jaw. Excessive local heat over area. *Chest:* Dullness in right middle third of post-chest. Wheezing rals, sonorous and sibilant at right base. *Heart:* Sounds weak. *Abdomen:* Negative. *Extremities:* Negative. *Reflexes:* Not obtained.

Clinical Notes, June 14: Temperature varied between 105° and 104°. Pulse was between 118-126. Respiration, Cheyne-Stokes, 22-28. Blood pressure: systolic, 132; diastolic, 80.

On Admission: Temperature, 104° F. Pulse, 120. Respiration, Cheyne-Stokes. Pulse bounding. Face is flushed and complains of pain in throat. Irrational, latter attempts to get out of bed. Unable to swallow, pulse more bounding and rapid, respiration shallow, drowsy and at times comatose.

June 15: Condition worse. Swelling on the right side of neck larger, very red and extends down to the clavicle on right side. Hypodermoclysis given. Throat painted with 50 per cent of neosalvarsan in glycerin; 2:33 p. m., ceased to breathe.

Laboratory Examinations: X-ray of Chest: There is a diffuse dilatation of the aorta. There is no evidence of consolidation of either lung. *Urine:* Specific gravity, 1018; acid, amber, albumin, ++; glucose, 0; acetone, 0; moderate number of granular casts, few W. B. C. *Blood Count:* Red blood cells, 3,700,000; W. B. C., 2,400; polymorphonuclear leukocytes, 0; mononuclear leukocytes, 50. Only 50 cells were counted. *Blood Chemistry:* Glucose, 080; urea N., 14.0; N. P. N., 30.0; creatinin, 1.5. *Blood Culture:* Not reported. *Throat Culture:* Few streptococci, pneumococci.

Therapy: Hypodermoclysis, 450 c.c. of Ringer's solution given twice. Transfusion: 350 c.c. blood. No immediate reaction. Neosalvarsan in glycerin, 50 per cent, local application to throat.

Comment and Conclusions: Agranulocytic angina is a symptom-complex more frequently diagnosed today than previously, due to our having become more thoroughly acquainted with the symptoms, course and general clinical picture of this condition, through the publication and frequent reports of these cases in medical literature.

Routine blood counts and throat cultures should be done in all cases of sore throat where the diagnosis is in question, particularly where the pathology is obscure and where there is associated infiltration of the tissues in the mouth or pharynx. In all severe cases of angina a blood culture should be taken as well. All cases of suspected Vincent's angina should have a blood count done in order to ascertain the blood picture for purposes of making a differential diagnosis.

The case here reported is considered a typical case of agranulocytic angina for the following reasons: *1.* Sudden onset with rapidly increasing prostration and high temperature. *2.* Severe sore throat (angina). *3.* Physical findings in the throat. *4.* Typical blood count (agranulocytosis). *5.* Rapid progress of disease to fatal termination.

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277 East Broadway.

REPORT OF A CASE OF LUDWIG'S ANGINA.*

DR. EDGAR J. STEIN, Lancaster, Pa.

My chief reason for wishing to report this case is that it was so thoroughly typical that it could hardly have been mistaken for anything else.

A man, C. G., age 72 years, came to see me, Oct. 29, 1928, to have his nose and throat cleaned out as he felt he was getting a cold and his family physician had advised him to go home to bed for a day or two in order to clear up his condition quickly. He was of the robust type, with excellent habits, so I gave him the usual cleansing and applied antiseptics to his nose and throat. At this time there was no indication of the impending trouble and I could see nothing out of the usual in his instance. In fact, I had entirely dismissed him from my mind when his family physician called me, three days later, and asked me to see him and see if I could not open his "quinsy" as he seemed to be suffering a good deal and had difficulty in breathing. This last statement caught my attention particularly, so that I got to see him as quickly as possible.

The patient presented a very striking chain of symptoms. He spoke with a very guttural voice and complained mostly of difficulty in breathing and a sensation of his throat closing, and said it had been impossible for him to swallow for the past 24 hours. Upon examination, I found that he could not open his mouth very wide, and when he did open it, the tongue appeared to take up all the room there was. I managed to depress the tongue sufficiently to see the fauces and in so doing I found that the left half of the tongue appeared to be hard and could not be depressed and was very painful on pressure.

The tongue appeared to be pushed up very much and there was a convexity in the floor of the mouth instead of the usual hollow. On palpating this I found it quite hard and indurated and quite tender. There was a submental swelling or fullness about an inch to the left side of the median line, with practically no tenderness on touch. The fauces appeared negative, with no indication of quinsy.

I immediately admitted him to the hospital and under general anesthesia—nitrous oxid and oxygen—made an incision through the

*Read before the Philadelphia Laryngological Society, April 2, 1929.

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skin and superficial tissues, and then with a pointed hemostat I went through the muscle tissue and into the abscess cavity about 2½ inches from the skin surface. About 1 ounce of thick extremely foul pus was evacuated and the track of the hemostat widened out by removing the instrument with the jaws open, and then inserted two fenestrated rubber tube drains. The anesthetic was extremely difficult because of the swelling in the pharynx and only by using an airway could we maintain respiration. His temperature on admission was 99.1°, which rose to 100.2° after operation and to 101.1° the second day, after which it gradually receded until it became normal on the fourth day in hospital.

The pulse never went above 90, and for the most of his stay was from 88 to 72. Respirations were mostly at 20. There was nothing of interest in the blood and urine examinations and a culture of the pus was reported as containing staphlococci, micrococcus catarrhalis and a fusiform bacillus (probably Vincent's).

Patient was discharged after a stay in the hospital of 12 days, with practically a completely closed incision, and has remained well since then.

While in the hospital a molar on the lower left side was troubling him and this was removed on the tenth day under novocain by his dentist, with no further trouble.

In looking back over the case several facts are brought out very forcibly. First, the extreme rapidity of development; second, the marked difficulty in breathing; and third, the anatomical changes about the sublingual space.

Blassingame (Arch. of Otolaryngology, Aug., 1928, Vol. VIII, pp. 159-176) gives a most excellent description of the anatomy, as well as the pathology of this disease, and also a very complete bibliography.

There is no doubt in my mind that the origin of the infection was the bad molar on the left side, which extended through the lymphatics to the sublingual space.

611 Woolworth Building.

PERITONSILLAR ABSCESS, THROMBOSIS OF THE EXTERNAL JUGULAR VEIN, AND BRAIN ABSCESS.

DR. HENRY M. GOODYEAR, Cincinnati.

The seriousness of a peritonsillar abscess, commonly called quinsy, is often disregarded except for the unusual discomfort of the patient Yet Newcomb reported a series of 41 cases followed by hemorrhage and 23 deaths. Fatal cases have also been reported by Carmody, Mosher, Coggin, King, Lange and others.

Recently there have been six fatalities in this city, and some time ago I reported a fatal case before our local Ear, Nose and Throat Society. The case which I now present is that of a young man, age 18 years, who was referred to my office complaining of pain across the frontal and occipital regions (both sides) and some tenderness over the right side of the neck.

The patient appeared very ill, and was sent immediately to the hospital. There was a history of having had a sore throat three weeks before, at which time a peritonsillar abscess had been drained on the right side. The tonsils were now of moderate size, not inflamed, and the abscess had apparently healed.

On close examination a partial paralysis of the right side of the face was evident. The right corneal reflex was absent and there was considerable pain and difficulty in bending the head forward. All other reflexes were normal. The patient had lost considerable weight, but there had been no vomiting. A vertical nystagmus of the right eye suggested a subtentorial irritation. The eye grounds were normal. The spinal fluid was normal and the white blood count was 26,000. Roentgenograms of the sinuses and mastoids revealed no pathology.

A thrombosis of the right internal jugular vein was suspected, with some intercranial lesion. However, on opening the neck a large abscess was found, together with a thrombosis of the external jugular vein. The internal jugular appeared to be normal and blood drawn from this vessel for culture was sterile.

A marked swelling occurred in the right upper orbit and lid two days later, but incision yielded no pus. A beginning cavernous sinus thrombosis was thought of, but after several days the swelling of the eye subsided.

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Five days after entering the hospital, it was noted that over 200 ounces of urine was passed daily without glycosuria, while the intake of fluids was about 85 ounces. An irritation of the pituitary body was suspected and was verified later at autopsy. The general septic condition prevailed and gentian violet was given intravenously; also 5 c.c. of the blood from the patient was injected intramuscularly.

Thirteen days after entering the hospital, a right hemiplegia developed, with unconsciousness. At this time Dr. Carter explored the left frontal lobe, but no abscess was found. However, the left lateral ventricle was markedly compressed to the left. A suboccipital decompression was done immediately and the right cerebellum was found covered with free pus.

Two days later the patient died and on autopsy an abscess 3 c.m. in diameter and 1 c.m. deep was found immediately over the pituitary body. There was no evidence of thrombosis in the cavernous or other venous sinuses. The right internal jugular was not thrombosed. A general suppurative meningitis was evident.

On looking through some 30 references preparatory to reporting this case, I found a number of cases of thrombosis of the internal jugular vein and brain complications had been reported. However, there was no report found pertaining to an involvement of the external jugular. That this vein in this particular case should be thrombosed without an extension into the internal jugular was rather difficult to understand.

The vertical nystagmus and the marked polyuria were of unusual interest among the symptoms of intracranial complications.

556 Doctors Building.

SOME OF THE COMPLICATIONS OF LARYNGECTOMY.

DR. J. E. MACKENTY, New York.

There are a few operators in this field who would like to convey to the world at large the consoling idea that by some special method of their own devising, laryngectomy has been reduced to a simple procedure. One of these optimists told me last year that he had just accomplished his 100th operation without a death or a complication. But since he had prevaricated on other occasions I was neither impressed by his skill nor depressed by my own comparative lack of it. It would take a rare combination of circumstances and a lot of good luck to pilot one through 100 laryngectomies without trouble.

I shall speak on the miseries which have come to me in this work and say a few words on their cause, prevention and treatment.

Hemorrhage: The foreign statistics make considerable mention of postoperative hemorrhage as a complication, even attributing to it a material death rate. Postoperative hemorrhage has occurred only once in about 300 operations, and this patient was a bleeder. Transfusion saved his life. Our technique practically obviates the possibility of hemorrhage. All vessels clamped are tied by transfixion. The patient is out of the anesthetic before the wound is entirely closed. If straining starts any bleeding it is readily seen and controlled, as the wound is open.

Embolism: Five cases of embolism have occurred in my series—four of them fatal—and all in patients between 75 and 80, excepting one of 63. In one autopsy obtained (a syphilitic) we found numerous old infarcts in the organs, a fresh clot in the right brachial and in the right cerebral arteries. Two died of intracranial lesion, after about three months of what was first thought to be a psychosis. The terminal stage of one was meningitis and of the other, focal softening. The so-called psychoses in the postoperative old are, I believe, frequently due to emboli into the brain.

Blocking of the Right or Left Main Bronchus or Both Main Bronchi with Inspissated Secretion: Years ago I discovered this complication by investigating the bronchial tree with a bronchoscope in a patient almost moribund from what was then thought to be pneumonia. The removal of a large plug of inspissated secretion from

*Read before the New York Academy of Medicine, Section on Laryngology and Rhinology, May 27. 1929.

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both main bronchi saved his life. This occurs often enough to be kept in mind when there is present any embarrassment in breathing.

Uremia: In the very old, a careful watch on the blood chemistry and meticulous dietetic adjustments are imperative to ward off uremia. This past season a man of 81 survived the operation after passing close to uremic coma. A daily check up after operation by a metabolist and careful metabolization before operation determined his recovery.

Diabetes: Diabetes and cancer seem to be in some way related. A high sugar index is frequent in cancer patients. In all such, a careful postoperative watch is kept on the blood sugar. Healing may be delayed and the incidence of infection much increased by a moderately high blood sugar, not true diabetes. I believe that small doses of insulin during the convalescent period are helpful in this condition. In true diabetes the dangers are great. Without insulin and the help of an expert metabolist we could not save any of these patients. Apart from delayed healing and wound infection, the great majority of diabetics now come through. From four to 12 weeks is the usual course. I believe that the tendency to recurrence is increased by diabetes.

Radium and X-ray: Of all the miseries that beset us, perhaps the most hopeless come from the indiscriminate use of radiation. After operation a dry gangrene attacks the whole radiated area, making hypopharyngeal closure impossible. Plastics fail in 100 per cent. For these cases, impossible to close, I have devised an artificial esophagus, described in a paper read before the American Laryngological Society in 1928, soon to be published in the Archives of Otolaryngology.

When radiation is combined with diabetes we are not justified in operating. Recently I was induced, against my better judgment, to operate such a case. Seven weeks after operation, gas gangrene attacked the neck and chest. This was controlled. Patient died four months after operation from gas gangrene.

Hiccough: This may occur after any surgical procedure but I think it is more frequent after laryngectomy. In 300 operations it has manifested itself about 15 times. It has not proved fatal in any case. In one it persisted off and on for five months. I can add nothing to its causation. Its treatment is anything from tickling the soles of the feet to pulling out hairs from the crown of the head, all equally efficacious in some hands and equally futile in others.

Tuberculosis of Lungs adds a great risk to the operation. The wound usually becomes tuberculous. In one patient with syphilis, tuberculosis and cancer of the larynx and active T.B. in the lungs,

the tracheal stump sloughed for over an inch, T.B. attacked the fascia of the neck, causing extensive undermining of the flaps; granulations failed to appear; the wound broke down entirely. In 12 months the neck had healed, leaving a slit opening into the hypopharynx. Excepting for this and his T.B. in the lungs, the patient has fully recovered. With the aid of the artificial esophagus, he can swallow his saliva and soft foods.

Pneumonia: The bugbear of the earlier operators has occurred only twice in my experience. The patients were very old and the termination was death. The type was bronchopneumonia. This low incidence of pneumonia in the one-stage operation contrasts very favorably with the statistics in the multiple-stage operation. New, at Mayo's, reports three deaths from pneumonia in 42 cases following the first stage but no deaths following the other stages. The explanation of this, I believe, is to be found in the surgical procedure employed. In the first stage the larynx and part of the trachea are skeltonized and the wound closed with a small drain. This, please observe, is a clean wound and no general anesthetic is used. Why this high death rate from bronchopneumonia? I believe that it is due to severing the laryngeal nerves, particularly the superior laryngeals. Anesthesia of the larynx ensues. The safeguard of normal sensation being removed, food and saliva find their way into the lungs. This danger does not occur in the one-stage operation.

Mediastinitis, formerly a seven-headed dragon to the older surgeons, has not occurred in my series. It should be exceedingly rare if the principles of the operative and after-technique are carefully observed.

Plastic Repair following failure of the esophagus and hypopharynx to close is now very rarely necessary. Our failures to secure complete closure, in the first operation, have occurred only in radium injury, T.B. and diabetes.

Laryngectomy, it must not be forgotten, is in the majority of instances done on patients in the declining years of life, and sometimes in the period of senility. All complications incident to this age may occur. In my series, the surgical mortality between the ages of 75 and 81 (the oldest patient operated) has been about 50 per cent in a 3 per cent total mortality; it is evident that surgical death is rare in the younger patients. This 3 per cent mortality occurred almost entirely in patients over 63. Laryngectomy has therefore taken its place among the comparatively safe major operations, with perhaps a few more complications to be guarded against than are usually met with in other major surgical procedures.

111 East 61st Street.

BRANCHIOGENETIC CYST OF LARYNX REMOVED BY THYROTOMY.*

DR. CHARLES J. IMPERATORI, New York.

M. M. T., female, age 51 years; occupation, telephone operator. Patient first seen Sept. 25, 1928.

Past History: Complained of asthma for 20 years. Within the past seven years there has been a change in voice and the patient felt that following a nasal operation done some few years ago, her hoarseness started. It has only been of recent date that she has complained of coughing and choking sensations. There has been no expectoration whatsoever and she has been able to carry on her occupation until recently. There is also a history of having had some type of growth removed from the region of Stenson's duct, the diagnosis having been that of a calculus or an abscess, the patient is not certain which. At all events, the operation was not entirely successful, for at times there has been a breaking down of tissue and a discharge of pus in the mouth. There is no evidence of any salivary fistula.

Family History: Slightly suggestive, in that a sister has an enlargement in her right neck that has been very slowly but progressively increasing in size for a period of some 15 years. This mass has given the sister no particular discomfort, excepting for the disfigurement incident to a growth in the neck. This enlargement is located in the region of the anterior border of the sterno-cleido-mastoid muscle.

Predominating symptoms were that of a stenosis of the larynx due to a large mass located within it and including the arytenoid and aryepiglottic fold and occupying over two-thirds of the airway on the right side. It was such that the epiglottis was pushed considerably to the left. There was no glandular involvement, and no fistula or pitting of the skin in the region of the right neck. The patient had lost some weight and her appetite was not so good as it had been. There was no temperature or chills.

Biopsy was suggested before removal.

Tentative Diagnosis: Nonmalignant tumor and that it was either a dermoid, enchondroma or a cyst.

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*Read before the New York Academy of Medicine, Section of Laryngology and Rhinology, Jan. 23, 1929.

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Biopsy was done Sept. 27, 1928.

Report of Biopsy: Paraffin sections showed edematous mucous membrane showing marked chronic inflammatory reaction in the deeper parts. There was no evidence of malignancy in the sections examined.

This report was somewhat unsatisfactory and it was decided that inasmuch as the patient had such difficulty in breathing, that the growth be removed by thyrotomy.

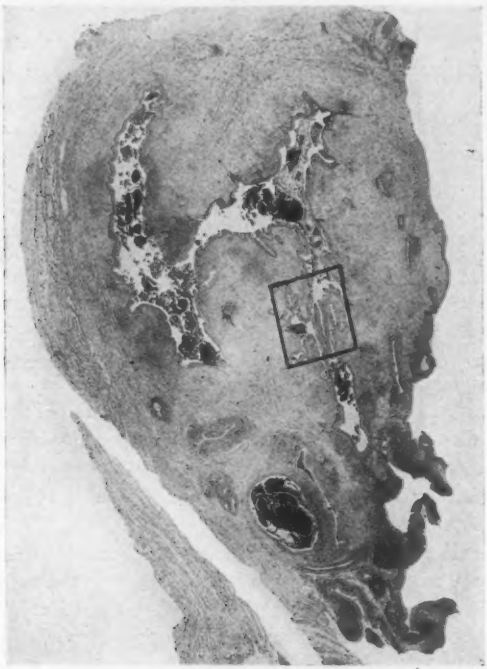

Fig. 1. Multilocular branchial-cleft cyst of larynx. Photomicrograph low power.

This was done on Oct. 1, 1928, under general anesthesia in the usual way, excepting that part of the thyroid cartilage was resected with the growth and a portion of the arytenoid was also removed. The operation was preceded by a tracheotomy. The tracheotomy tube was removed, Oct. 3, and the patient was fed through a nasal feeding tube until Oct. 10. The tracheal wound was closed by Oct. 13 and the thyrotomy wound was closed on Oct. 20.

Patient made an uneventful recovery.

There has been no recurrence of new tissue in the region of the growth and her asthmatic symptoms have entirely disappeared. She has a fair speaking voice. Examination of her larynx at this time shows an adventitious band stretching across the upper entrance of the larynx on the right side and a vocal cord on the opposite side. The amount of the deformity of the larynx is that seen following the removal of a vocal cord and adjacent tissue.

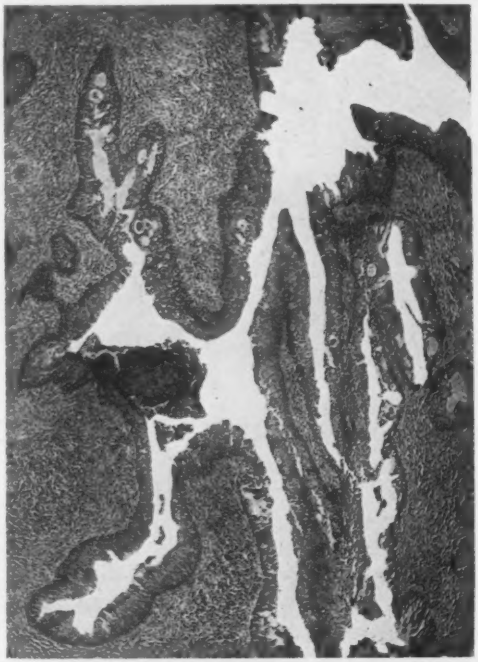

Fig. 2. High power of area included in marked square in Fig. 1.

The growth was 42x28x18 m.m. and it was an irregularly lobulated mass that was fairly soft on pressure, with firmer portions in the deeper parts. The cut surface resembled fibrous tissue in which there were several round openings suggesting ducts. The tissue about these openings were cartilaginous in consistency in some areas.

Microscopically, the tissue showed canals lined by pseudo-stratified columnar epithelium typical of the respiratory tract. Most of the

canals were filled with plugs and necrotic epithelium and these plugs were filled with gritty, calcific granules.

Diagnosis: Multilocular branchiogenetic cyst with chronic inflammation of its wall. This was the report made by Dr. Meeker, Associate Pathologist at the New York Post-Graduate Hospital.

This type of growth is rare.

Lateral cervical cysts and fistula result from improper obliteration of the second branchial cleft. As a rule the structure of these cysts or fistula is constant. Their lining consists of stratified columnar epithelium, varying at different points in the number of cells. No celia are present. Beneath the epithelium there is more or less an unbroken layer of lymphoid tissue. Surrounding this is a supporting layer made up of strands of fibrous tissue rich in areola connective tissue spaces of large sizes. These support the network of arterials

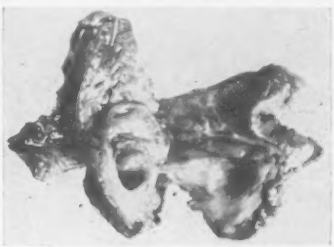

Fig. 3. Multilocular branchial-cleft cyst of larynx.

and capillaries. A more or less viscid fluid is given off from the lining.[1]

It might be of note to mention that there was some granulation tissue located in the anterior commissure until a month ago. This is rather a common occurrence following a thyrotomy and particularly where the cartilage has been partially removed. Recently the granulation has entirely disappeared and at present there is a small spicule of cartilage that is extruding itself and can be very easily seen. This also is common where an extensive operation is done in this region.

There is a great temptation to remove this granulation or cauterize it. It should be left alone.

Mention is made of this so that those who operate upon a patient, doing a thyrotomy and remove a malignant growth, have fair reason to feel that what might appear to be an immediate recurrence is granulation tissue. This observation has been confirmed many times.

A few cases are reported in the literature, one by Dr. Coakley[2] in

1904, and another by Dr. Harris[8] in 1908. This latter growth was removed through a laryngoscope and necessarily must have been of small size.

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17 East 38th Street.

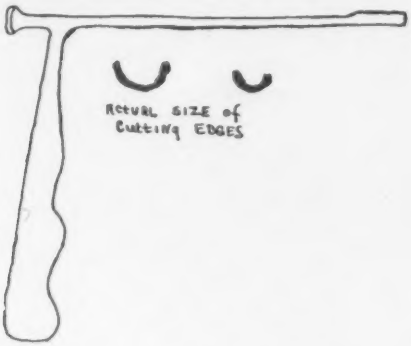
A SPHENOID GOUGE.

DR. LOUIS K. GUGGENHEIM, St. Louis.

The anterior wall of the sphenoid sinus should be removed in its entirety in such cases as give indication for surgical treatment. Superiorly the anterior wall is usually quite thin and may be broken down with a Hajek hook, a Sluder knife, a nasal forcep or almost anything else that will reach. Inferiorly the anterior wall always shows an increase in thickness. In some cases this inferior portion is so thick as to be impossible of removal with anything other than a chisel or a burr. The writer used a burr once. Chisels have all been unsatisfactory because every chisel, heretofore on the market, has necessitated two strokes to remove an appreciable amount of bone. The difficulty this has presented is that the second cut could rarely be made at the proper angle to the first cut to remove the amount of bone desired; particularly in cases of profuse bleeding. The sphenoid biting forceps have all been disappointing in cases with a very thick anterior wall. They slip off the bone or they nibble. Many cases in the past have been sent out of the operating room with a

goodly ledge of anterior wall remaining because it seemed impossible to remove it. The writer has broken off a Sluder knife in the sphenoid and had a most embarrassing time getting it out, so the use of this instrument in the thick portion of anterior wall is to be discouraged. The giant magnet is a good thing to remember should one have such an accident.

The thought occurred, about three years ago, that a sphenoid chisel, to be really efficient, would have to be so constructed as to remove



with one stroke a chunk of bone, regardless of thickness of the anterior wall. Such a chisel had to be grooved, and sufficiently deeply grooved, to accomplish the task.

The instrument, illustrated, was made in two sizes by V. Mueller and Co.

It has not only proven a great comfort in sphenoid work but has been almost equally useful in removing the most anterior portion of the lateral nasal wall in antrum window resection.

The grooved end is so shaped that with one stroke the anterior end of the window can be completed, whereas with the Wagner forceps, other biting forceps and chisel, many efforts are necessary to accomplish this part of the operation.

1000 Carleton Building.

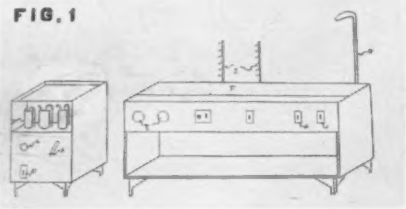
A NEW BRONCHOSCOPIC INSTRUMENT TABLE.

Dr. Simon Jesberg, Los Angeles.

I wish to present a bronchoscopic instrument table which I have successfully used for over one year. The table as shown is divided into two units: A suction stand and the table proper. This arrangement makes it possible to use the suction stand as an independent unit in nose and throat surgery. However, when the table is to be used solely for bronchoscopic work, both units are better combined under one top.

The suction lines are controlled by a two-way valve. This device makes it possible to aspirate secretions either into the right or left suction jar or into both simultaneously. The change is made instantly by moving the lever (A) into any of the desired positions. No shifting of rubber connections is necessary. This valve has

FIG. 1



been in use for over four years and has never been out of order mechanically.

As shown here the suction apparatus is run from a central plant. However, there is no reason why a portable suction pump could not be inserted in the suction stand.

The third jar (B) is connected to the ether blower. A light placed below this jar makes it possible to easily determine the amount of air passing through the ether and at the same time serves in a slight degree to keep the ether jar warm.

The rheostats which control the lights for the bronchoscopic instruments are shown at E. The dry batteries are placed within the table.

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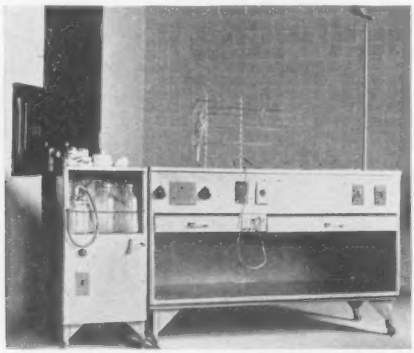
The top of the table proper is made in two sections. The front one-third is made of sheet metal and the rear two-thirds (F) of frosted heavy plate glass. A battery of four lights, controlled by switch (G) is placed below this glass. By means of this glass and the lights it is possible to silhouette the instruments against the sterile sheet which covers the table. We feel that this feature is valuable since it makes it less difficult for the nurse to pick out the desired instrument in the darkened room.

The light (H) is for the purpose of illuminating the room or the top of the table. This light is controlled by the switch (I).

The instrument racks (J) are self-explanatory and have been used for a number of years in a modified form. These racks (J) fit into slots and can be instantly removed for sterilization.



Besides the features described, the table contains a number of convenience outlets which allow for the plugging in of cautery or other electrical equipment.

By allowing the sterile table cover to fall over the front of the table and thereby covering the switches, it is possible for the nurse to manipulate the switches without contaminating her hands.

The table has been found helpful in facilitating the work in the Bronchoscopic Clinic of the Eye and Ear Hospital of Los Angeles. It was designed for me by J. J. Cantor, whose advertisement appears in this issue.

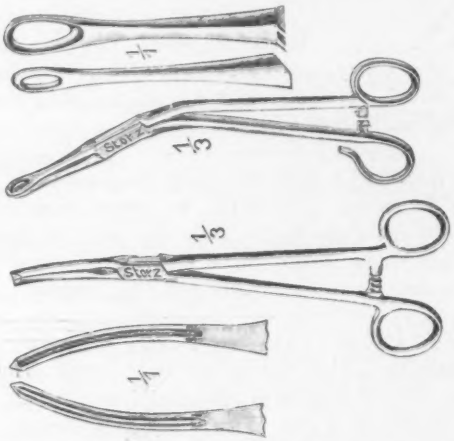
500 South Lucas Avenue.

NEW INSTRUMENTS.

Dr. Albin M. Painter, Kansas City, Mo.

Forcep No. 1. Tonsil grasping forceps. As shown in the illustration, the under jaw is smaller, giving more friction upon the tonsil mass, as it crowds it partially through larger fenestra. The larger blade comes in contact with the capsule after partial dissection has been made and it is found to have a good retaining grasp on the mass, allowing sure and careful manipulation in stripping the tonsil from its bed, as the other forcep, acting as a hemostat, clamps the vessels within their fascial covering. The larger blade protrudes beyond the smaller, making grasping easier, as well as more effective.

Forcep No. 2. Hemostatic forceps of angiotribe type, using the principle of the Ferguson forcep. These may be used as scissors in



dissecting, though instead of cutting, it clamps the tissue with a firm, lasting grip, while the mass is stripped away. The vessels thus are found to be more effectively clamped than by the usual serrated-edged forceps. The groove and tongued blade do not allow slipping. The approximal surface is so ground that the tissue is not cut, but severed at the distal surface, leaving crushed vessels when forceps are removed. These hemostatic forceps grasping the fascia as it is stretched from the bed, instead of cutting as with scissors as the dissection progresses, will leave a dry fossa and less danger of postoperative hemorrhages.

These forceps are manufactured by C. R. Storz, Indianapolis, whose advertisement appears in this issue.

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International Digest of Current Otolaryngology.

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Dr. D. E. Staunton Wishart, Toronto.
St. Louis Jewish Hospital E.N.T. Journal Club.

The Middle Section of the American Laryngological, Rhinological and Otological Society announces that its next meeting will be held Tuesday, Jan. 21, 1930, in Rochester, Minn. Dr. Ross H. Skillern, president of the society, will be present. Those desiring further information should correspond with Dr. H. I. Lillie, chairman of the Middle Section, Rochester, Minn.

We are in receipt of a news item from Czechoslovakia, reporting the death of Professor O. Kutvirt, for many years a leader in Czechoslovakian otolaryngology. Only last year he founded and edited Otolaryngologia Slavica. The new editor of this journal is Professor Dr. Ant. Precechtel, Durdikova 603, Prague.

The American College of Physical Therapy announces its Eighth Annual Meeting, to be held Nov. 4, 5, 6 and 7, 1929, at Hotel Sherman, Chicago. The Society is divided into three Sections, of which Ear, Nose and Throat is one. Papers in physio-therapy, in all of its ramifications, are to be presented. Further information can be obtained from Dr. F. L. Wahrer, Secretary, Marshalltown, Iowa.

Kuhns, in the Archives of Pediatrics, 46, 34-40, 1929, discusses the treatment of bronchial asthma. He considers asthma as a toxemia due to change of body metabolism and believes that this toxemia produces a bronchial spasm when the nasal mucous membrane has been irritated. He also believes that the rhinologist must clear up all infections in the nose before the bronchial affection can be improved. M. F.

Liebermann, in the International Zentrallblat fur Ohrenheilkunde 29, 1928, reports on a case of gonorrheal arthritis of the arytenoid joints. The case was one of polyarthritis of gonorrheal origin and

affected the arytenoideal joint, producing a swelling of the surrounding structures with fixation of the vocal cord. Healing occurred after treatment with X-ray and diathermy. The author points out that until his case there had only been eleven such cases reported and mentions that most of the other cases were misdiagnosed as laryngitis.

M. F.

It is rather interesting to note that the trend of treatment in certain conditions of otolaryngology is apparently on the up-swing of the usual pendulum.

In the September, 1929, Journal of Laryngology and Ootology, Cleminson reports on the treatment of malignancies of the esophagus by thoracotomy and implantation of radon seeds. His results as yet do not show any startling results except for the fact that the patient does get some immediate relief.

Immediately following this article in the same journal is one by Woodman, who uses the esophagoscope to insert radon seeds. His results also show some advancement in the treatment of malignancies of the esophagus.

In this same journal in the Society Proceedings of the Royal Society of Medicine, Section on Otology, there are numerous discussions on the treatment of chronic deafness and the striking point of all the discussion is the swing to the use of diathermy, zinc ionization and other electro-mechanical appliances.

The users are among the foremost otologists of England and when they, as a group, report in their Society some striking results of these newer methods it becomes obvious that the time is not far off when the pendulum swing shall have reached its center and some actual, concrete therapeutic information will have been gained.

M. F.

The Kansas City Society of Ophthalmology and Otolaryngology has issued its program for 1929-30. Monthly meetings are scheduled and many interesting conferences have been arranged. Numerous visitors have been invited to read papers at these conferences and undoubtedly the Society is going to have a very successful year. Additional information concerning these meetings may be obtained from Dr. A. E. Eubank, 636 Argyle building, Kansas City, Mo.

Dean, of St. Louis, read a paper on "Nasal Sinus Infections in Children", before the Section of Laryngology, Rhinology and Otology of the American Medical Association at the Portland meeting in

July. The paper appears in the September 14 issue of the Journal, American Medical Association. Dean brings out some very important facts, based on his long experience, which are very interesting. He claims that latent sinusitis is far commoner than heretofore believed. He states that infection of the sinuses in children is often overlooked because the infection may have little clinical significance. He has found that a chronic sinusitis very often occurs with some systemic disturbance, such as asthma or arthritis, and by the clearing up of the sinusitis the general systemic disturbance is alleviated. Dean is of the opinion that diet and hygiene are especially important in dealing with sinusitis in children and believes that when tonsils, adenoids and allergy have been ruled out, diet is the next most important factor. He quotes Daniels and Marriott in the influence that vitamin A plays in nutrition in sinusitis in children. The treatment is both pediatric and laryngologic and both must be used in common as neither is sufficient in itself. M. F.

Our Italian colleagues have suffered the loss of four great leaders of Italian otolaryngology. Massei, Gradenigo, Ferreri and Grazzi have all died within a year and otolaryngologists in Italy have been dealt a hard blow. The memory of these four great men will stir otolaryngologists, not only in Italy but throughout the world, to greater achievements. M. F.

Dixon, of Cleveland, in the Sept. 14, 1929, issue of the Journal, American Medical Association, reports on the occurrence of nasal septum perforations in chromium workers.

It seems that workers in this industry are prone to nasal perforations and lesions of the skin. Dixon studied eighteen cases of perforation of the nasal septum in one industrial plant and on examining the surroundings he found that the trouble was due not only to the presence of chromium but that ventilation played a major part. In other establishments where identical work was being done, but under good ventilation, he found no cases of nasal perforation. The action of chromic acid is such that it fixes the tissues by coagulation (Beck).

Perforation of the septum occurred in such a manner that unless one was aware of the possibility of chromic acid irritation, lues or tuberculosis would be blamed. Dixon draws the conclusion that ventilation in the industrial plant using chromic acid can protect the workers and that a bland oil spray serves to protect the nasal mucous membrane. M. F.

Cemach, at the December, 1928, meeting of the Vienna Laryngo-Rhinological Society, presented a case showing an extraordinary course of a chronic ethmoiditis with total destruction of the ethmoid, which extended up to the dura but with absolutely no signs of pus. The patient was 43 years old and in June, 1928, complained of severe frontal headache which had lasted over a year, coming on every now and then at least every two or three weeks. Apart from this, there was some hindrance to nasal breathing, no snuffles, nasal secretion was normal and never of a yellow color. Examination revealed a very excessive septum deviation which was corrected by operation. Following this the patient claimed that she felt absolutely well but in August of the same year (1928), she again reported that her headaches had returned within the last three weeks and seemed to be localized over the left frontal region. Objectively there was a large swelling of the left middle turbinate; there was nothing else to see. An interesting finding was that the patient had a very high-grade cocain idiosyncrasy and instead of the mucous membrane shrinking under cocain and adrenalin it became hyperanemic and turgescent, filling the entire nasal cavity. No pus at any time was seen in the nose and the patient stated that she never had to use a handkerchief. X-ray examination revealed an absolutely normal picture; the left ethmoid region showed no change.

Because of the unbearable headaches, the middle turbinate was resected and an endonasal ethmoid attempted. The ethmoidal labyrinth was completely filled with granulations but there is no pus. Because it was decided that a larger communication with the base of the skull should be made, the operation was continued from the external route and the large exposure showed complete destruction of the sphenoidal and ethmoidal labyrinth. The destruction of the ethmoid extended to the dura mater.

Histological examination of the resected material gave no definite information; Wassermann reaction was negative. The patient made an uneventful recovery and left the hospital six days after the operation and within three weeks her headaches disappeared completely and did not return.

In the discussion of this presentation several cases of cocain idiosyncrasy were presented and Professor Hajek asked if it was not possible that this condition may have been an extension into the nose from above rather than a nasal condition extending upward.

M. F.

BOOK REVIEWS.

The Surgical Treatment of Goitre. By Willard Bartlett, A.B., A.M., M.D., D.Sc., F.A.C.S., St. Louis, with foreword by Dr. Charles H. Mayo, Rochester, Minn. With 130 Illustrations. St. Louis: The C. V. Mosby Company, 1926. Price $8.50.

A well written work which embodies all surgical phases of goitre. The pathology, indications for operation and technique are heavily stressed. The book is profusely illustrated with interesting figures. A chapter is included on the relationship of the heart to goitre, and a chapter on laryngeal complications of goitre. The book is of interest to surgeon and laryngologist alike and because of its literature references, it makes a useful acquisition to all libraries.

M. F.

Goitre Prevention and Thyroid Protection. By Israel Bram, M.D. Author of "Goitre; Non-Surgical Types and Treatment"; Medical Director, Bram Goitre Institute, Upland, Pa.; formerly Instructor in Clinical Medicine, Jefferson Medical College, Philadelphia, etc. Illustrated. Philadelphia: F. A. Davis Company, Publishers. 1928.

Dr. Bram has written this book in a style which will help the layman understand some of the problems of the thyroid gland.

The book is of value to the student and practitioner, but is not detailed sufficiently to be of great value to the surgeon or endocrinologist. M. F.

Diseases of the Larynx, including those of the Trachea, Large Bronchi and Esophagus. By Harold Barwell, M.D., London; F.R.C.S., England; Consulting Surgeon of Diseases of the Throat and Ear, St. George's Hospital, etc. Third Edition. Humphrey Milford, Oxford University Press. 1928.

This small volume on the Larynx, Trachea and Esophagus makes a very useful reference book not only for the specialist but also for the general practitioner. It is written in a clear, concise manner and discusses the diagnosis and treatment of several laryngeal conditions in a manner that facilitates the actual handling of the patient. Prescriptions are appended and the technique of various operations is outlined. There is a chapter on Intubation and one on Laryngotomy which clearly explain these emergency operations. The book can well be recommended to anyone interested. M. F.

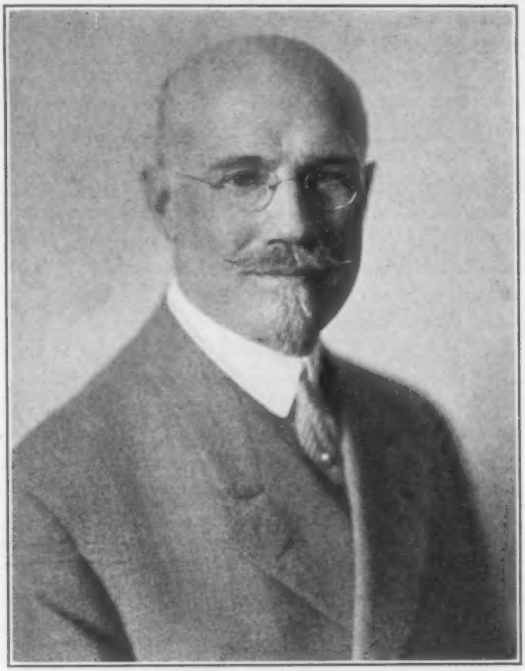
Lehrbuch der Lokalanästhesie. By Dr. Caesar Hirsch, Chefarzt der Abteilung fur Hals-Nasen und Ohrenkranke des Marienhospitals in Stuttgart. With foreword by Professor Dr. O. Voss, Direktor der Universitatsohrenklinik in Frankfurt a/M. With 104 illustrations. Stuttgart: Verlag von Ferdinand Enke, 1925.

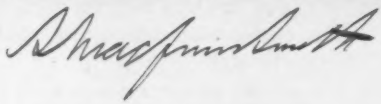
This is one of the few textbooks in any language which deals with specialized local anesthesia. Professor Otto Voss, in a foreword, says that he feels that Hirsch's book fulfills one of the largest gaps in Otology, Rhinology and Laryngology. The book itself systematically takes up the question of local anesthesia, its origin and its evolution up to the present time. The chemical changes are worked out with formulae to prove the reactions.

The nervous mechanism of pain is graphically demonstrated so that the rationale of local anesthesia can be the better understood. The technique of each operation and local anesthesia, from the neck up, is clearly outlined and Hirsch deserves much credit for having banded together such important information into one volume. The book is richly illustrated to show the anatomical and technical points. M. F.

In Memoriam

Another of the "Old Guard" in American Oto-Laryngology, Dr. Seth MacCuen Smith, has passed to the Great Beyond. His death occurred Sept. 15, 1929, preceded several days by a severe attack of angina pectoris. He was 66 years old.





SETH MacCUEN SMITH.

A native of Pennsylvania, he was born in Blair County in 1863; he was graduated from Hollidaysburg Academy and received his medical degree from Jefferson Medical College, in 1884. In 1889 he was married to Miss Virginia Allen, of Germantown, who, with three children, George Allen Smith, Mrs. Stuart L. Bullivant and Lewis MacCuen Smith, survives him.

When a department of oto-laryngology was established at the Germantown Hospital, Dr. Smith was placed in charge and since that time has been extensively engaged in the intensive study of these specialties in surgery.

In 1904 he was appointed Professor of Otology at Jefferson Medical College; he also was Attending Otologist at Jefferson Hospital and functioned actively at the Germantown, Jewish, Memorial and Oncologic Hospitals.

When the American edition of the "Atlas and Epitome of Otology" was being brought out he was selected as its editor. He wrote many treatises on the diseases in which he specialized. He wrote a chapter on "The Treatment of Diseases of the Ear and Tympanic Membrane" in the volume "Modern Treatment and American and English Authorities", and was the author of a chapter, "Diseases of the Middle Ear" in the volume, "Sajous' Analytic Cyclopedia of Practical Medicine". In addition he was the author of many contributions to various medical journals.

During the World War Dr. Smith was commissioned Major in the Medical Reserve Corps. He did some notable work in laryngology with cases of soldiers whose throats had been corroded or otherwise injured by poison gases.

Dr. Smith was a Fellow of the American College of Surgeons and of the American Medical Association, a member of the College of Physicians of Philadelphia, the Medical Society of the State of Pennsylvania, the American Otological Society, the American Laryngological, Rhinological and Otological Society, of which he was President in 1916, and the American Academy of Ophthalmology and Otolaryngology.

As a recognition of his long and valuable service to American Otology he was named President-Elect of the American Otological Society in June, 1929, and had assumed the honorary and active duties of office in this distinguished organization in preparation of its next Annual Meeting in May, 1930.

His personality was genial, gentle and gracious; he was broad-minded, far-sighted and definite in his scientific spirit and attitude. In his discussions before national otological societies his position, justified by his wide and varied experience, was authoritative and forceful and he was always accorded a respectful hearing. He stood for the best in medicine and always reached for the higher ideals in manhood; he was a humanitarian, an earnest scientist and a well-bred gentleman.

His family life was ideal and to his sorrowing wife and children we offer our sincere sympathy. M. A. G.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTOLOGY.

April 12, 1929.

Surgical Anatomy of the Temporal Bone; Lantern Demonstration. Dr. George E. Shambaugh.

Dr. George E. Shambaugh discussed surgical anatomy of the temporal bone, showing lantern slides illustrating these relations. He pointed out that in the preparation for practice in a specialty, one of the most difficult problems which the student has to solve is the problem of acquiring that sort of knowledge of the intricate anatomy of the temporal bone which would make it possible for him to carry out various operative procedures with a minimum of risk of injuring important structures. The work in anatomy which is provided for under graduate medical students does not provide the facilities for acquiring this knowledge. To take operative courses where various operations are repeated also fails to provide that knowledge of surgical relations, because as one after another of these relations are exposed the progress in operation alters or destroys the picture.

In order to get the facts fixed in one's mind it is necessary that preparations be made with the view of bringing out this or that relation, and bringing it out in such a way that it is easy to grasp and fix in one's mind. In order to make such preparations it is necessary that one devotes thought and care. To make sections through the temporal bone, sagital, frontal and horizontal without an effort to emphasize clearly this or that important relation is of very little value. Such sections will, of course, bring to light each and every anatomical structure, but in such a manner that they are of very little assistance for building up that background of anatomical relations which the student requires, who undertakes to operate in this region.

Dr. Shambaugh stated that from his own experience in preparing himself for special practice the acquiring of practical knowledge of the surgical relations was found to be more difficult than that of acquiring the clinical background necessary for evaluating one's findings in examining patients.

It is not feasible to describe the series of lantern slides which were exhibited. Among these was demonstrated a series of temporal bones of the developing child, beginning at birth when the absence of the mastoid process, of the bony external meatus and the presence of a petulent petrosquamosal suture was pointed out. Of the adult temporal bone, he showed a series illustrating various types of surface configuration and how the linea temporalis was not that constant structure which some textbooks take it to be in using it as a landmark for operating upon the antrum. In some instances it takes an abrupt turn upwards immediately back of the external meatus; in others, it courses downwards to about the middle of the external meatus before taking its horizontal course backwards. In either of these cases operating through the so-called triangle in an effort to reach the antrum would lead to disaster.

Various types of mastoid processes were shown illustrating the differences in pneumatization from the completely pneumatized process where the pneumatization has been completely arrested. He pointed out the difference between a partial or complete arrested pneumatization and the condition met with where there has been a chronic suppurative otitis media; that is, the condition of osteosclerosis where pneumatized spaces have been entirely obliterated. Confusion regarding these two types of processes has lead to some faulty deductions regarding the clinical importance of the absence of pneumatization.

Arrested pneumatization seems to be due, in most instances at least to the development in early childhood of some tubotympanic inflammatory process, which may, or may not, be suppurative in character but which retards or prevents the development of normal pneumatization. It is a pretty well recognized clinical fact that this type of process is more of a menace than a completely

pneumatized process when in subsequent years such an individual becomes the victim of acute purulent otitis media, as there is much greater danger from complications only partly due to the thickened cortex, which obscures the condition that is developing within the process but also to the fact that a bone invading process seems much more likely to develop in the individuals with arrested pneumatization.

In the mastoid process showing the absence of pneumatization as the result of osteosclerosis secondary to the long-standing suppurative otitis media, the condition is quite a different one, as osteosclerosis seems to be Nature's method of obliterating the disease. This it is able to do in most instances, but in those cases where, because of the type of marginal perforation, there develops an extension of epidermis unto the middle ear cavities, the cholesteatoma thus formed not only prevents the disease from healing but in itself constitutes one of the most serious menaces that we encounter in chronic suppurative otitis media. Now, strangely enough, some have put the matter in an entirely opposite sequence and consider the sclerosing of the mastoid processes a condition which leads to the chronic suppuration, especially in those cases with cholesteatoma.

A series of lantern slides were demonstrated showing the surgical relations of the tympanum and the relation of the facial canal to the external auditory meatus. The development of tympanic cells and of tubal cells and the clinical significance of these anatomical variations was discerned.

DISCUSSION.

DR. E. B. DENCH: I feel it a privilege to speak on this occasion. I appreciate most keenly the splendid work Dr. Shambaugh has done in this line, and I am glad that he has brought before us a paper so full of scientific work and so eminently characteristic of himself, and yet one that possibly I may discuss with a little intelligence.

In the first place, a point that interested me very much was Dr. Shambaugh's remark in regard to locating the mastoid antrum. Although I have read McEwen's book a number of times, I have never yet been able to map out his triangle, although he has given it as a landmark for the antrum. I agree with Dr. Shambaugh that that is useless. I have, however, a triangle of my own, and I think it locates the mastoid antrum very well. I have never found it to fail; if you take a line tangent to the superior wall of the meatus and another line tangent to the posterior wall, the triangle formed by these two lines and including the curvilinear superoposterior border of the meatus, which lies between the two lines, always lies over the mastoid antrum and an artificial opening within this space will enter the cavity. Sometimes the superoposterior border of the meatus slopes considerably. This indicates that the sinus far forward and that the antrum is to be opened through this sloping area.

What Dr. Shambaugh said about the jugular bulb arising in the floor of the tympanic space is perfectly true. On one occasion I wounded the bulb in this region, but after applying a packing of iodoform gauze to the spot, and then grafting the remaining portion of the cavity, I found when I removed the pledgets and the strip of iodoform gauze, that the entire cavity had dermatized perfectly. In a second case, the dome of the jugular lay in the floor of the middle ear and encroached upon the posterior tympanic space. Sharp hemorrhage occurred, which was early controlled by a packing of iodoform gauze. When the packing was removed the hemorrhage had ceased, so I lined the entire cavity with a primary skin graft, taking care that the graft was held firmly in position by the packing, where it covered the wound in the bulb. In this case also the cavity dermatized perfectly. Anyone who operates a great deal will occasionally wound the dome of the jugular. If great care is taken, however, to clean out the septic cavity thoroughly before the final steps of the operation are completed in this region, it has been my experience that injury to the jugular dome does not add much to the danger of the operation. I have never lost a case of this kind.

I was very glad to hear what Dr. Shambaugh had to say about the sclerotic mastoid: that it was in most cases a result of chronic suppuration and not the cause of it. This is in opposition to the view of Cheatle, who holds that the infantile type of bone is the cause of suppuration. I agree thoroughly with Dr. Shambaugh.

I also agree with the speaker regarding the danger of the sclerosed mastoid. I have always felt that a bone of this type, in a case of acute inflammation, is the worst bone with which we have to deal.

The sections showing the course of the facial nerve through the mastoid were interesting. In Dr. Shambaugh's sections the nerve seemed to be located at a considerable distance behind the posterior boundary of the tympanic cavity. This is not always the case, however, and very frequently the nerve passes close to the posterior wall of the middle ear, and may be easily wounded in exposing the posterior tympanic space. It should always be remembered that in some cases the nerve curves forward beneath the floor of the external auditory meatus, and is sometimes exposed in this region in lowering the floor of the canal to obliterate the hypotympanic space. I have exposed the nerve three or four times in this region without any untoward results.

I was also delighted with what Dr. Shambaugh had to say about discharge coming from the Eustachian tube after the radical operation and the statement that such discharge was absolutely devoid of danger to the patient. While all of us aim to obtain a dry ear in every case of radical operation, in cases where the tubal cells are very well developed it may be impossible to actually close the tube, by applying the skin graft, or by any other method with which I am acquainted. I have tried a bone graft in this region in one case with success, but I do not employ it as a routine procedure. It is well to explain to the patients before the radical operation that the operation will relieve them as far as danger to life is concerned, but that the promise of a dry ear cannot be made in every instance, although this has occurred in over 90 per cent of my cases. By an explanation to the patient that a mucus discharge may persist after the operation, but that in no instance is it a menace to life, we place ourselves in a much better position as far as the patient is concerned. It is the discharge which annoys the patient, and for which he seeks relief. The explanation to him that the operation is done primarily to save his life and that the continuation of a small amount of mucus discharge is to be looked upon as merely an annoyance will do much to effect a better understanding between the patient and the surgeon.

DR. H. B. BLACKWELL: This subject, so ably presented by Dr. Shambaugh, is one of great practical importance to the otologist. I, too, feel that the characteristic anatomy usually found in the mastoid in the presence of chronic suppuration, e. g., the infantile type, with far forward sinus, low overhanging dura, high antrum, with practically no cells present save in the antrum region, is the result of chronic suppuration developing during infancy and childhood and is not produced by congenital malformation. Such appearances are rarely found when the suppuration develops after adolescent life. Chronic suppuration of the ear extending during infancy and childhood results in a pinched-in or infantile appearance of the mastoid, which I find analogous to the facial appearance of an adult who during the same period of life suffered from post-nasal obstruction caused by adenoids. Both are developmental anomalies, due essentially to faulty pneumatization of the affected parts.

Two other points shown by the pictures are: first, the relatively large size of the epitympanum, which is a space quite as large, if not larger than the middle ear itself; and second, the wedge-shaped bone which constitutes the postero-superior auditory canal wall and at the same time the external wall of the attic or epitympanum and which prevents direct drainage from the antral region in chronic suppuration. As most of those present probably know, I have for a number of years felt that the removal, partial or complete, of this wedge will permit direct drainage from the mastoid in cases of chronic mastoiditis and result in a cure without exenteration of the middle ear structures.

DR. L. T. LEWALD: I would like to know if Dr. Shambaugh has seen a case with congenital absence of the ear and external auditory meatus. I have just been asked to X-ray a child to see whether in trying to form an external ear it would be proper to attempt to form an external meatus. As Dr. Shambaugh has just told us, it is very difficult to demonstrate any bony external canal in the newborn, and I have been unable to answer positively the question as to whether there was an external canal in a child of a few months of age. On the opposite side, the ear is well developed and normal.

DR. E. M. JOSEPHSON: I would like to question Dr. Shambaugh about the comparative frequency of radical mastoid operations in recent experiences as

compared with its frequency in the past. Also, what Dr. Shambaugh's experience has been with the medical treatment of these chronic running ears. In view of the pathologic anatomy of the chronic mastoids which he has shown us, it appears obvious that the abscess cavity and the active disease process in these cases is very frequently sharply localized to the region of the antrum and the upper part of the mastoid process. It seems but to be expected that a majority of these cases can be successfully treated by medical means, and without subjecting the patient to the risks of a radical mastoid operation, which view has been borne out by my own experiences.

DR. ALFRED BRAUN: I would like to ask Dr. Shambaugh to describe briefly his method of preparation of his histological specimens. I was very much interested in the case he described of injury to the internal carotid artery during the radical mastoid operation. It recalled to my mind a patient in the Manhattan Eye, Ear and Throat Hospital some 15 years ago; a case in which there was no injury at the time of operation; but three weeks later, while the patient was being dressed in the clinic, the house surgeon took out the packing and go ta gush of blood that shot across the room. With great difficulty he finally controlled the bleeding by packing. The patient was admitted to the hospital, and two or three weeks later developed headache, and after several more weeks went into coma, and had an attack of Jacksonian epilepsy, beginning in the arm of the opposite side. He was operated on and a collection of pus was found directly beneath the dura over the temporosphenoidal lobe, 3 or 4 dr. of pust being evacuated. He died the following day, and at autopsy the followlowing condition was found. There was a dehiscence in the inner wall of the bony Eustachian tube about a quarter of an inch in diameter. The carotid artery was uncovered in this location. The packing had been forced into the Eustachian tube and produced an area of necrosis in the wall of the carotid artery. This necrosis had caused the hemorrhage. A thrombus had formed at this point. A piece of the thrombus broke off and lodged in a terminal branch of the anterior cerebral artery. It produced an abscess in the frontal lobe of the brain about an inch-and-a-half in diameter. This perforated through on to the surface of the brain, and the pus gravitated backward in the subdural space until it reached the temporosphenoidal lobe. It was this subdural collection of pus which was tapped at the operation.

DR. SHAMBAUGH: The question was raised concerning the relationship between nondevelopment in the pneumatization of mastoid and the tubotympanic processes resulting from tonsil and adenoid disease. There seems to be very good reasons for believing that in long-standing tubotympanic processes suppurative, as well as nonsuppurative, may be a factor in arresting pneumatization in a young child.

There was a question asked regarding the substituting of some simpler operative measures for the radical mastoid in the treatment of suppurative otitis media. I would point out that it is only a few of the cases of chronic suppurative otitis media even where there is a persistence of discharge where operative measures are justified. These are the few cases where, in connection with the discharge, there is evidence of a bone-invading disease which constitutes a serious menace. In these cases the simpler operative measures which have been suggested to replace the radical mastoid are of very little assistance in removing the diseased focus.

An experienced otologist can usually make, even at a single examination, a differentiation between the common type of chronic suppurative otitis media where there is no bone-invading disease, and where local measures alone are applicable, and those exceptional cases where, associated with the discharge, there is a bone-invading disease. In the former case, operative measures are hardly justified. In the latter case, operative measures other than the radical mastoid, are of no avail. Ossiculectomy has been, more or less, extensively practiced as a substitute for the radical mastoid but only those cases are cured by ossiculectomy where operative measures are not indicated.

Another question came up in the discussion regarding the presence of a primary cholestatoma in the external meatus. Usually the cholesteatoma is associated with a chronic discharging ear, but conditions may arise which will bring about the piling up of desquamatic material in the external meatus, producing a veritable cholesteatoma. I have seen several such cases where I was satisfied there had never been an otitis media.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

Regular Meeting, Tuesday, April 2, 1929.

Interesting Case of Lateral Sinus Thrombosis. Dr. Henry Wieder.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. MACKENZIE: The case report presented by Dr. Wieder should not be allowed to pass without discussion, which is due him. This paper is in keeping with the other excellent papers that Dr. Wieder has presented in the past. While listening to the presentation of the paper several thoughts came to my mind. First, those of you who were present at the meeting of the American Laryngological, Rhinological and Otological Society in 1925 will recall the discussion of Dr. Frazier on sinus thrombosis, when he sighted the fact that the last five cases he saw managed to pull through without an operation, and Dr. Ewing Day, who followed him, spoke in a similar manner. I had the temerity to challenge both these gentlemen, not on their experience, but merely to bring before the attention of the younger men present that they must not accept these discussions as an endorsement of an overly conservative attitude in this class of cases.

I was called to see a case this last week in consultation with a very capable ear man, who had operated a patient five days before. It was a case of bilateral middle ear involvement and the doctor had operated the right side. I suggested that we operate the left side and we found the expected complication on that side.

The question Dr. Wieder asked himself: Is it well to go far when you do not get bleeding? This brings to my mind a case of primary thrombosis of the superior petrosal sinus. At the operation I removed the thrombus from the entire length of the sinus, but got no free bleeding from the cavernous sinus. The patient did exceptionally well for five days and my associate, who was looking after the case after the operation, told me that he thought the patient would recover; however, I felt very doubtful, realizing that I had not removed that part of the thrombus that extended into the cavernous sinus. About the sixth day, the patient's temperature rose and from then on the patient presented all the classical symptoms of cavernous sinus thrombosis with exitus.

Many times we depend upon blood cultures to determine the diagnosis. In the majority of cases we are disappointed by obtaining a negative culture in spite of a bloodstream infection. The bacteria are especially easy to obtain during a chill when there has been a fresh shower of bacteria swept into the general circulation and between the attacks the blood culture may be negative.

I have been using Pregle's iodin solution for a long long and prefer it to mercurichrome, which has a more limited use. We obtain the special iodin solution put up in ampules for the purpose and we use it in liberal quantity, as much as 20 c.c. at a time and at 24 or 36-hour intervals.

As to whether the thrombus beyond that part which we are able to remove is fibrous or infected, one cannot say. We have no right, therefore, to say that the rest of it was fibrous, although it is possible.

DR. COATES: I have not much to say except to compliment Dr. Wieder on the exceptional way he has presented his paper. It is a very interesting case and I agree with him in the procedure which he followed.

It is not necessary to go back to the end of the thrombus in order to get free bleeding. In some cases where I have had jugular bulb thrombosis, it has seemed safer not to attempt to clean out the bulb and unnecessary to tie the jugular vein.

Pregle's iodin is possibly the best of intravenous bacteriacides but my chief preference is for transfusion. Two years ago I had a child with sinus throm-

bosis, who was very rich indeed with a streptococcus hemolyticus infection. It so happened that in that case, I had operated on the mother for the same condition eight years before. At first I transfused with the father's blood, having forgotten the previous infection the mother had. A second necessary transfusion, made with the mother's blood, proved very much more successful, in fact, completely so. It seems quite possible that the immunity had lasted eight years. If you can get an immunized donor, it should be best, but I think transfusion is the best thing, whether you have an immunized donor or not.

DR. SHUSTER: The question, in operating on a case of sinus thrombosis, whether one is to try to get behind the clot or just open the sinus, leaving the back part of the clot alone, must often be decided at the operation. As Dr. Coates says, if great difficulties are met and the clot appears to be well organized, it is not necessary to attempt to get free bleeding. One case I recall in which I did get free bleeding, I had the opportunity to investigate post-mortem. This patient had also had a temporosphenoidal and a cortical brain abscess, to which he succumbed. Upon examination of the torcula, I found it full of pus. The infection had evidently extended backward in spite of the plug. My thought was at that time that it would have been better had I plugged the sinus well beyond the infection.

As to making up one's mind whether to open a sinus or not, I think that when one is really in doubt as to its contents and the trend of clinical signs point to a thrombus, the external appearance of the sinus not having a normal appearance, it is far safer to plug it above and below and investigate by incision. A needle puncture is not always reliable and the Ayer-Toby test has not been sufficiently reliable in my experience to rely upon it with assurance.

DR. WIEDER (in closing): In reference to Pregle's iodin and mercurochrome, we use Pregle's iodin in streptococcic infections and mercurochrome in staphylococcic infections.

Another thought has come to my mind concerning the Toby test. I feel that if you get a lack of rise in pressure over the diseased side, you can be sure of one thing, that your thrombus extends into the jugular bulb. If it did not, you should have some rise. If you get a complete blockage with no rise whatever, you can feel sure that the jugular bulb or inferior petrosal sinus, or both, are involved in the clot.

I was possibly influenced in my failure to go back to the torcula by Dr. Wayne Babcock. In his last series of cases, both he and Dr. Steele have tried doing nothing but incising the sinus and draining it, and they all got well. When he tried chasing the clot, he lost several patients.

Dr. Shuster just proves conclusively what this case shows. Even if you do get back and get free bleeding, you may have infection back of it, as shown in your post-mortem. If you cannot determine with a fair amount of satisfaction whether there is an obstruction in the sinus, while the patient is still on the operatin gtable, turn him over and try the Toby test while he is still under anesthesia. If the pressures are equal there is no obstruction.

Case Report of Ludwig's Angina. Dr. Edgar J. Stein

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. MACKENZIE: Was a blood sugar test made in your case? My reason for asking this question is that the last case I saw was one in which the patient's blood sugar was 266 mg. per 100 c.c. This patient showed marked signs of improvement only after attention was paid to the diabetes, but an expert internist who was called in consultation.

DR. STEIN (in closing): The blood sugar test was not made. The thing I did not stress was the odor connected with it. At the time of operation, the anesthetist nearly passed out from the odor. The odor continued for about five days. You must use rubber tubing instead of gauze in these abscesses. It is even difficult to keep rubber tubing in place.

Pyemia Following the Anginas. Dr. David Nussbaum.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Meeting of February 18, 1929.

Dr. Herschel Ezell, Chairman, Presiding.

Report of Bronchoscopic and Esophagoscopic Work for the Year 1928.

Dr. Hilliard Wood and Dr. W. W. Wilkerson.

The number of patients treated in this series was 29; 25 of whom gave a reasonably definite history of having swallowed a foreign body. In 19 a foreign body was found and removed. In six no foreign body was found, and in four cases the examination was for diagnostic purposes.

The 19 Foreign Bodies Found and Removed: Of the 19 patients in whom a foreign body was found and removed, the youngest was 11 months old, four were from 1 to 2 years old, four from 2 to 3 years old, three from 3 to 4 years old, two from 4 to 5 years old, one from 5 to 6 years old, one from 7 to 9 years old, and four from 9 to 60 years old. It will thus be seen that 15 of the 19 patients were under 8 years of age, or 78.9 per cent.

There were 12 females and seven males. The time the foreign bodies were retained varied from 1½ hours to 10 weeks. Six of the foreign bodies were retained less than 24 hours; in four cases the foreign body was retained between 24 and 48 hours; two were retained between 48 and 72 hours, two were retained four days, one was retained five days, three were retained 10 days, and one 10 weeks. Leaving out of consideration the foreign body retained 10 weeks, the other 18 cases showed an average time in which the foreign body was retained three days.

The foreign bodies were located as follows: Esophagus, 8; nose, 1; left bronchus, 1; right bronchus, 5; and pharynx, 4.

The foreign bodies found and removed were: Three beans, two watch wheels, three straight pins, one button, one thimble, two nickels, one fish bone, one quarter, one tack, one peach stem, two pennies and one horseshoe nail.

Ether was used in 12 cases, cocain in two cases, and no anesthesia in five cases.

Six Cases Who Gave a History of Having Swallowed a Foreign Body, in Whom no Body Was Found: There were six patients who gave a more or less definite history of having swallowed a foreign body, but in whom no foreign body could be found either by physical, X-ray or bronchoesophagoscopic examination. In some of these doubtless a foreign body had been swallowed, but possibly coughed up, or passed into the stomach before the examination was made. It is of especial interest that two of these patients, in whom no foreign body could be found, died. One, a child, who gave a history of having swallowed a grain of corn, but in whom no foreign body was found by bronchoscopy; and an adult male, who gave a history of having swallowed a piece of oyster shell, but in whom no foreign body was found by peroral endoscopy. From this it will be seen that failure to find a foreign body does not in any sense eliminate the danger of death.

Four of the patients had endoscopy for diagnostic purposes without any reference to a foreign body.

Fatalities: There were three fatalities. In one, a watch wheel was removed from the esophagus of an infant. The operation was done under ether. The next morning, during very inclement weather, the family carried the patient some 50 miles home. Four days after the operation the patient developed pneumonia and died on the sixth day. One was a child, who was supposed to have swallowed a grain of corn, but in whom no grain of corn was found. One was an adult, who was supposed to have swallowed an oyster shell, but nothing was found, and 12 days later the patient died in another city from some unknown cause.

DR. W. W. WILKERSON: As Dr. Wood stated, we had three deaths during the past year. It is our opinion that the deaths were not directly due to bronchoesophagoscopic work, but rather to things over which the endoscopist had no control. One of these cases died away from the city, due presumably to the trauma produced by the foreign body, rather than to any trauma which was produced by the esophageal speculum.

A second case had a tracheotomy and died because the tube was allowed to become plugged with mucus by an inexperienced nurse. The third case died from pneumonia some six days after the foriegn body was removed from the lung. Therefore, after understanding the causative factors of the deaths enumerated above, I feel that this is a very excellent report.

There was one case which we had during the past year which was of considerable interest, that of a child with a horseshoe nail in his lung. The point was in the trachea and the head down in the left bronchus. This case was examined both by Dr. Wood and myself and we were unable to locate the nail by means of a bronchoscope, even though the X-ray findings clearly and accurately showed its location. It was necessary for us to pass a bronchoscope under a fluoroscopic screen in order to visualize and remove the nail. While this is strange and certainly does not occur often, we had this experience during the past year and neither of us were able to account for the occurrence.

Meeting of March 18, 1929.

Dr. Herschel Ezell, Chairman, Presided.

Purulent Infection of Both Maxillary Sinuses. Dr. M. M. Cullom.

Mrs. T. A. M., age 62 years, consulted me on Jan. 26, 1929, complaining of neuralgia in right side of face. Transillumination showed both antra dark. Patient gave a history of having had both antra drained and frontal sinus "rasped out" last year. No opening found in either antra. X-ray picture showed both antra cloudy, all other sinuses clear.

Under local anesthesia at St. Thomas Hospital, a large window resection of the nasoantral wall was done under the inferior turbinate on both sides, Jan. 31. Irrigation brought away pus from both antra for something like 10 days, gradually decreasing in amount until the washings were practically clear.

Patient continued to complain of neuralgia on the right side of face. All her teeth had been extracted, but on account of the symptoms I told her brother, who is a dentist, that there might be a tooth root left. X-ray pictures disclosed a very large root in the lower left jaw, which I asked him to extract. He found an infected root, which was extracted with considerable difficulty. The next day there was a profuse discharge of pus from both antra, which continued for something like a week. The neuralgic pain promptly disappeared with the extraction of the root.

There are two distinct points of interest in this case, the neuralgic pain complained of in the right side of the face, which was relieved by the extraction of an infected root on the left lower jaw. The other point is, that the extraction was followed by intense lighting up of the purulent process in both antra. Was the tooth root the original focus of infection of the maxillary sinuses, or was the exacerbation of the purulent process a mere reaction to the traumatism and infection resulting from the extraction of the root?

Traumatic Mastoiditis. Dr. Eugene Orr and Dr. W. G. Kennon.

C. B., age 38 years, colored, laborer. Two weeks before consulting us, some molten iron dropped into patient's left ear. There was a purulent discharge and some foreign material, apparently oxidized iron, removed from the ear. This probably amounted to about 4 or 5 gr. in weight. He was treated with the usual routine for acute otitis media. After about a week or ten days' treatment, he developed all the symptoms of mastoiditis, and was sent to the hospital for operation.

X-ray of mastoids showed definite destruction on the left side. At operation, a cortica lperforation was found in the upper portion. The mastoid cells were

found full of pus and there was considerable softening of the bone; the usual mastoidectomy was done.

Post-operatively, the course was rather stormy for a time. At one time it was thought that he had a lateral sinus thrombosis; however, blood cultures were negative, and there were some signs in the chest, which may have explained the fever. He was discharged from the hospital 26 days after operation, and during a short subsequent period, dressings were done at the office.

Miss G. G., age 28 years, telegraph operator. First consulted us Feb. 14, 1929. Complaint: Pain in left ear since Jan. 24, 1929. While brushing her hair, a hair pin in some way was forced into the external auditory canal, and she was told it had ruptured the drum. The ear discharged for several days and then stopped, but the pain in the ear continued. The drum was reopened on Feb. 7, 1929, but though the ear discharged, there was no relief of pain and for a week the mastoid area was acutely tender. The signs and symptoms of mastoiditis were so typical that no X-ray was made.

She was admitted to the hospital and operated upon on Feb. 15, 1929. Leukocytes, 10,200. At operation, the cells were found full of pus. The bone was softened. There was a large extradural abscess and a rise of temperature to 102.6° on the day following operation. Her subsequent convalescence was uneventful and satisfactory. She was discharged from the hospital 10 days after operation.

DISCUSSION.

DR. M. M. CULLOM: I have seen a great many ruptures caused by trauma, but I have never seen one develop into a mastoiditis. I think that it is remarkable to have had two.

DR. EUGENE ORR: One thing that Dr. Kennon did not mention is that in both cases the discharge was of a granular appearance. The negro boy discharged longer and more than any mastoid I ever saw.

Meeting of April 15, 1929.

Dr. Herschel Ezell, Chairman, Presided.

Open Safety Pin Removed from Stomach. Dr. Hilliard Wood.

April 13, 1929, about 10:30 a. m., patient swallowed an open safety pin. W. D. N., age 12 months, was taken to Vanderbilt Hospital, where an X-ray picture showed an open safety pin in the upper esophagus, the open end up and the sharp point directed towards the left.

At 1 p. m., examination with fluoroscope showed open safety pin in the stomach. The pin was removed in the following manner:

Using a Jackson laryngeal speculum, the Wood safety pin forceps were passed through the esophagus into the stomach. By the aid of the fluoroscope the pin was maneuvered into a position where the crotch of the pin could be included in the ring of the forceps. The pin was simply drawn up and out, with the open end down. The removal of the pin was done without anesthesia, local or general, without the esophagoscope, and without any visible blood. Patient put to bed in good condition. This pin was a small, gold safety pin, 20 m.m. long and, when closed 4 m.m. broad.

Fractured Larynx. Dr. Hilliard Wood.

Dr. Joseph G. Gray, Franklin, Ky., referred to me H. E. W., male, white, age 45 years, on March 18, 1929. The patient gave the following history: March 17, 1929, while cranking his Ford, it backfired and he struck the right side of the neck on the radiator cap, injuring his larynx. He was considerably stunned, and his voice was hoarse, but he could breathe normally. Patient sent to Nashville same evening and placed in St. Thomas Hospital.

Patient first seen by Dr. Wood in St. Thomas Hospital on March 18. His breathing was normal and his voice practically normal. There was some soreness about the larynx and the right side of the thyroid cartilage seemed somewhat depressed. 4 p. m.: Examination of the neck showed the larynx dis-

placed one-half inch or so, towards the left. Palpation showed some depression of the right wing of the thyroid cartilage. When the larynx was pushed back into the medium line the depression above mentioned practically disappeared.

Throat examined with laryngeal mirror. The right vocal cord appeared normal; the left vocal cord showed a very slight redness. In a general way, the larynx seemed normal and the cords moved normally, both in respiration and phonation.

Treatment: A gauze compress, held on by adhesive strips, was placed on the left side of the larynx in an effort to hold it in the middle line. This only measurably succeeded. As the patient's speech and breathing seemed practically normal, and he had only a slight soreness about the neck, he was allowed to return home and requested to report to his physician, Dr. Joseph G. Gray.

Dr. Gray, in a letter on April 4, says that the patient's voice has returned to normal, he is back at work, and his larynx is about one-half inch out of line.

A Case of Death Following Tonsillectomy. Dr. Herschel Ezell.

I am reporting this case under the above caption for the reason that I do not consider the patient's death due to the operation of tonsillectomy. The tonsil operation was only an incident, the complications of which were unquestionably the cause of death.

On April 10, 1929, Dr. J. W. T. Dabbs phoned me that he had a little girl patient, F. S., age 9 years, who had a severe case of acute articular rheumatism, who had developed a bad heart, both attributable to diseased tonsils. He stated that the child had been ill for 10 days with the heart trouble and rheumatism and that the temperature had ranged from 100° to 103° during that time and that he was anxious to have the tonsils removed at once. We both agreed that this was a hospital case and the patient was sent to St. Thomas Hospital the afternoon of April 10. The usual laboratory examinations were ordered made.

On arriving at the hospital that night I found a very sick patient; temperature, 102.6°, suffering greatly with many painful joints, both in the upper and lower extremities and the body. The pulse was rapid and irregular and I, although not an expert on the heart by any manner of means, could elicit a faint murmur. The tonsils were definitely septic. Urinalysis was negative, except an occasionally epithelial cell. W. B. C. was 10,200. Under these circumstances I advised against an immediate operation and the parents urged that it be done. They were told that the patient was too ill to undergo the operation, and with much persuasion they agreed to leave the patient in the hospital a day or so for further study.

On the following morning the patient's temperature was 100.4°, but otherwise the condition had not changed. We put her upon soda salicylate gr. V, T. I. D. Feeling that we were dealing with a very serious case prompted me to talk with Drs. Wood, Wilson, Tarpley, as well as with Dr. Dobbs. Dr. Wood and Dr. Wilson were of the opinion that the removal of the tonsils offered the best results. Dr. Wilson stated that it was safe to operate if the white blood count was under 14,000, regardless of the temperature. Dr. Wood stated that the operation should be done without delay and was the safest course to pursue. With everybody agreeing that we should go ahead with the operation, we decided to operate on the morning of April 12.

The usual preparation was given, viz., no breakfast and a hypodermic of morphin, gr. ⅛, 15 minutes before the operation. Ether was decided upon as the best anesthetic and Dr. J. R. Tarpley was engaged to administer it. The operation was performed by the snare and dissection method and the adenoid removed at the same time. There was very little bleeding and nothing happened to mar the success of the operation until the patient was ready to be moved off of the operating table, when she suddenly stopped breathing.

The patient's head was immediately lowered and artificial respiration begun. Dr. Murray Davis and Dr. Frank Dunklin volunteered their assistance and hypodermically. These measures failed to revive the patient and it looked like rendered all the help that they could. Digafoline was suggested and given the patient was dead. Adrenalin was suggested to be injected directly into the

heart, without avail. Despite everything we could do, the patient died. The cause of the patient's death in my opinion was the heart affection.

Sojous describes a condition known as heart-block that seems to fit this case precisely. The typical feature of this condition is marked slowing of the pulse. This condition often occurs, when it occurs at all, complicating acute articular rheumatism. Willus states that the striking objective feature in practically all cases is the lack of definition of the heart sounds. Sojous states that true heart-block is due to excessive activity of the adrenals and over-stimulation of the cardiac nodes and bundles. He states that adrenalin can only do harm and is contra-indicated. He advises amyl nitrite or nitroglycerin. I have been unable to find any comment upon the administration of ether in these cases, yet I feel that it is the safest anesthetic.

DR. HILLIARD WOOD: Did the patient get cyanotic, become choked?

DR. EZELL: No, her respiration was about normal, her color was good, and her general behavior was good.

DR. HILLIARD WOOD: Dr. Ezell telephoned me and gave me the outline and I advised an operation. I am very sorry of the result. I still don't know why the patient couldn't go ahead. I don't believe that the so-called chronic heart lesions have much to do with ether.

DR. ROBERT SULLIVAN: One-eighth gr. of morphin could kill a patient just like ¼ gr. could kill an adult. Morphin in a child will never be given by me.

Meeting of May 20, 1929.

In Absence of Chairman, Dr. Hilliard Wood Presided.

A Radical Operation on the Mastoid. Dr. M. M. Cullom.

Mrs. J. P., age 29 years, consulted me on April 18, 1929, in regard to her left ear. She was evidently suffering and her general appearance and bad color indicated that she was quite ill. She gave a history of chronic purulent discharge from the left ear, extending over a period of 15 years. In 1918, she had a polypus removed from the ear. Present illness dates back 10 weeks. During that time she has suffered with pain, nausea, vomiting and constant vertigo. A good part of that time has been spent in the hospital.

Inspection showed a profuse discharge of foul pus from the left ear. The canal was filled with polypus. There was no nystagmus. Compression of the tragas increased the vertigo, but produced no demonstrable nystagmus. There was no tenderness over the mastoid. With a Barany noise apparatus in right ear, only loud voice sounds could be distinguished. Her physician was advised of the gravity of her condition and an early operation was urged. A guarded prognosis was given and the danger of lighting up a meningitis was pointed out.

On April 22, 1929, the operation was performed at St. Thomas Hospital. The incision and exposure of the bony canal disclosed a perforation above the canal. The cortex was quite hard and was removed with mallet and gouge. I found that the entire posterior wall of the bony canal had been destroyed, just as if it had been removed by a radical operation. A large cavity was uncovered, which was filled with thick, cheesey pus of a very foul odor. There did not appear to be cholesteatoma, such as we would expect to find in such a case. There was an exposure of the dura above and a large exposure of the lateral sinus. There was a fistula in the external semicircular canal. I curetted the dead bone from the opening. I cautioned the anesthetist to watch the face carefully when I came to curette the middle ear. She reported twitching of face as soon as I reached this region. The facial nerve was lying exposed in the middle ear and by touching it with the curette I could produce twitching of the face. I cleaned the middle ear as thoroughly as I could. I hesitated to do the plastic and close the posterior wound in the presence of an exposed dura and lateral sinus, but finally decided to do so. I cleaned away all diseased tissue to the best of my ability and then filled the wound with alcohol and allowed it to soak for a while. I then did a Neumann flap and closed the external wound with Michel skin clips, leaving a small opening at the bottom of the wound for drainage.

Following the operation, there was a slight rise of pulse and temperature and the patient complained of vertigo for several days, but vomited only a few times. The wound was not dressed for four days. Considerable pus drained from the lower angle of the wound. The wound was dressed daily and alcohol was freely instilled. After a few days the drainage from the external wound ceased and the wound closed. The vertigo disappeared after about a week. The patient left the hospital in 10 days and is now apparently well. The vertigo has entirely disappeared, her color has greatly improved, she is sleeping and eating well and her physical appearance has undergone a great change.

Meeting of June 17, 1929.

Dr. Herschel Ezell, Chairman, Presiding.

Tumor of the Epiglottis. Dr. W. W. Wilkerson, Jr.

B. H. H., white, male, age 34 years, came into my office complaining of having had laryngitis three times within the past six weeks and giving no history of any peculiar sensation of fullness of his throat. The family and past history were essentially negative.

On examination, his tonsils showed very difinite signs of chronic inflammation. The nares and sinuses presented no pathology. An examination by his dentist revealed five abscessed teeth. A tumor about the size of a small marble was noted on the anterior surface of the epiglottis on the right side. It was pedunculated and had a broad base. It felt rather firm and showed no signs of infectious or degenerative process. No other pathology was noted in the larynx.

Under ether anesthesia the tonsils were removed, after which the patient was suspended with the Lynch apparatus. The growth on the epiglottis was removed with a snare and scissors. We first grasped the growth with a simple tenacula, which ruptured it, allowing approximately 1 c.c. of gelatinous material to escape. The snare was then passed around the collapsed mass, tightened sufficiently to hold it, and with the aid of scissors the tumor was freed from its base. The patient made an uneventful recovery.

Pathological examination by Dr. J. H. Litterer revealed the growth to be a simple cyst.

Complicated Mastoiditis. Dr. W. W. Wilkerson, Jr.

E. H. W., white, male, age 30 years, gave the history of having had pain and discharge from the left ear for the past five weeks. Infection in his left ear was preceded by acute coryza. The family and past history was essentially negative.

On examination, the right ear revealed no pathology. The left auricle was protruding forward and the canal was filled with pus. So much drooping was present that even after the canal was cleaned only a small portion of the drum could be seen. An X-ray examination revealed the cell walls to be completely broken down on the left side and a sclerotic condition on the right side. The nose and throat were substantially negative. No involvement of the sinuses could be made out. The blood count was 18,500, with 87 per cent polys. The temperature varied between 102° and 103°.

At the time of operation a subperiosteal abscess was found. Contrary to the X-ray report, only a few cells were present, while the greater portion of the mastoid area was sclerotic. Evidently the subperiosteal abscess had confused the Roentgenologist, as well as myself. Much free pus was present within the few cells and all of their walls were definitely broken down.

Upon uncovering the sinus we suspected it to be thrombosed, and upon opening it found this to be the case. We freed the sinus o fall its bloodclots until we obtained free bleeding from each direction. We then walled the sinus off with iodoform gauze.

Patient's temperature following the operation never rose above 100°. The iodoform packing over the sinus was removed on the eighth day and no bleeding ensued. The patient made an uneventful recovery and was discharged as cured 18 days later.